

ETS  
ISSUE BINDER

EXPOSURE  
A REVIEW  
OF THE LITERATURE

VOLUME I

2023380066

INCHES

1

2

3

4

5

6

7

8

9

10

SHEET LIFTER MUST BE ON TOP OF PAPER

2023380067



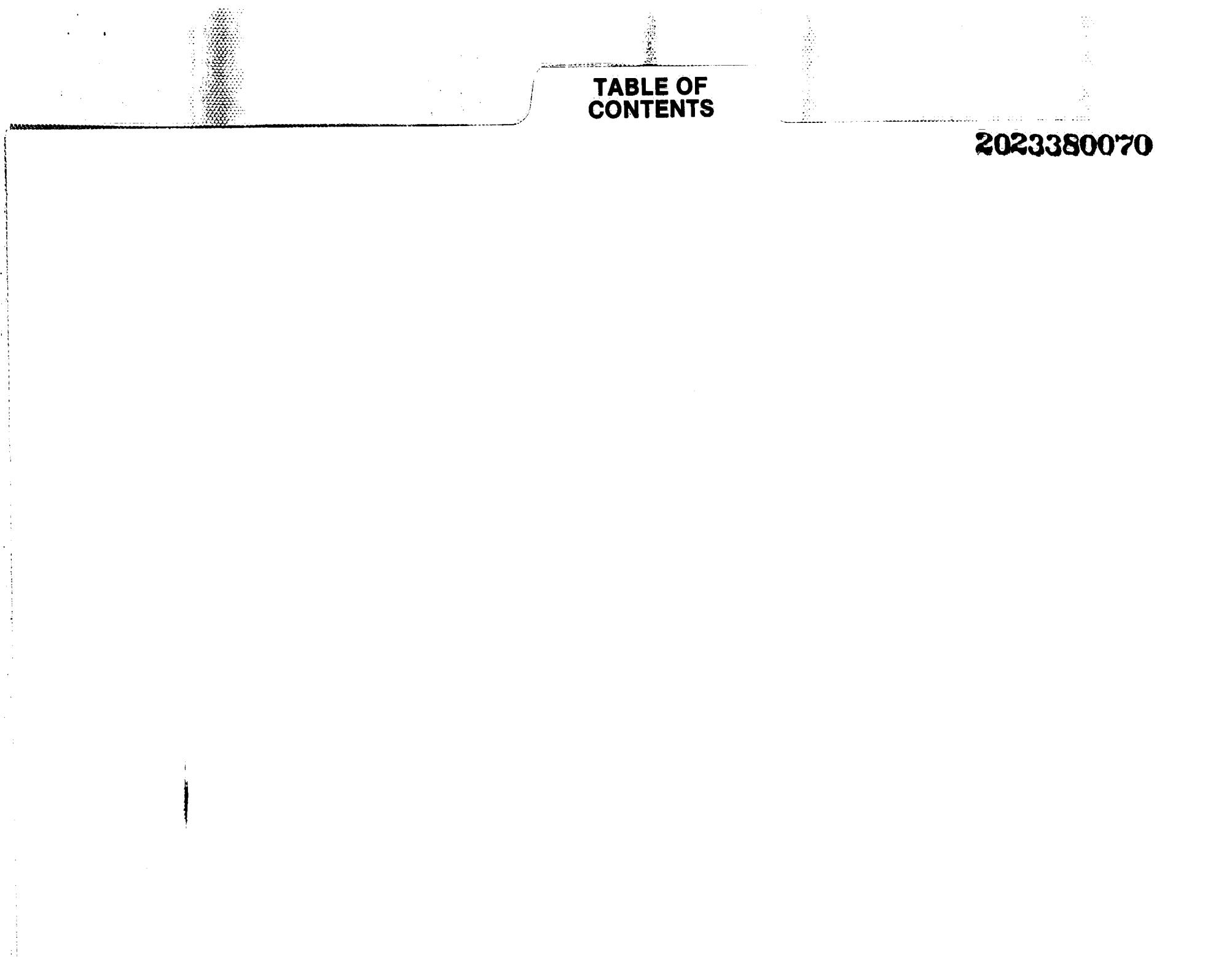
**ENVIRONMENTAL TOBACCO SMOKE  
EXPOSURE STUDIES  
A REVIEW OF THE LITERATURE**

**2023380068**

THIS ISSUE BINDER IS INTENDED TO PROVIDE A BASIC,  
COMPREHENSIVE REVIEW OF THE SCIENTIFIC LITERATURE  
REGARDING A SPECIFIC TOPIC ON ETS AND THE HEALTH OF  
NONSMOKERS.

PRIMARY STUDIES AND REVIEWS HAVE BEEN HIGHLIGHTED  
TO IDENTIFY (1) USEFUL OR HELPFUL INFORMATION (YELLOW  
HIGHLIGHT) AND (2) ADVERSE RESULTS OR OPINIONS (BLUE  
HIGHLIGHT).

2023380069



## TABLE OF CONTENTS

2023380070

TABLE OF CONTENTS

I.	ETS: A CHARACTERIZATION	1
	References	4
	Studies	A
II.	EXPOSURE TO ETS	5
	Questionnaire Reliability	7
	ETS and Radon	7
	References	9
	Studies	B
III.	DETERMINATION OF DOSE	13
	Cotinine	13
	References	14
	Studies	C
	Lung Retention	16
	References	17
	Studies	D
	Mutagens	18
	References	20
	Studies	E
	Other Biological Markers	21
	References	23
	Studies	F
IV.	BIOLOGICAL PLAUSIBILITY	24
	References	26
	Studies	G

2023380071

V. APPENDIX I . . . . .	27
Summary Review Article	
VI. APPENDIX II . . . . .	28
Aircraft Air Quality . . . . .	
References . . . . .	
VII. INDEX . . . . .	36

2023380022

**2023380073**

### ETS: A CHARACTERIZATION

- Environmental tobacco smoke (ETS) is an aged and dilute mixture of sidestream smoke (SS), or the smoke from the burning end of the cigarette, and exhaled mainstream smoke (MS), the smoke to which the smoker is exposed.
- ETS differs chemically and physically from both MS and SS. ETS is a dynamic, ever-changing mixture which, as it ages and dissipates, undergoes chemical reactions and physical change. There is no single definable, reproducibly characterizable entity known as ETS.
- Dissipative forces such as air currents and attraction to surfaces influence SS and exhaled MS. Studies indicate that constituents in ETS are hundreds to thousands of times more dilute than either SS or MS. Often, concentrations of ETS constituents fall below detection limits of current scientific measurement devices.
- As ETS ages, a number of physico-chemical changes take place. Matter evaporates from SS particles as they age to ETS. During the aging process, ETS particles coagulate and increase in size. Chemical compounds partition between the gas and

2023380074

particle phase of the smoke. (For example, nicotine is found in the particle phase of MS; in fresh SS, most of the nicotine is in the gas phase.) Decay patterns for constituents of ETS vary over time and are dependent upon physical conditions in the environment.

- ETS is not equivalent to either MS or SS. Many studies and reviews employ sidestream/mainstream smoke comparisons, ostensibly to demonstrate the kind and quantity of constituents involved in exposure to ETS. But such comparisons are deceptive and misleading. As two tobacco smoke chemists reported in 1990:<sup>1</sup>

Although ETS originates from sidestream and exhaled mainstream smoke, the great dilution and other changes which these smoke streams undergo as they form ETS make their properties significantly different from those of ETS. Thus, the sidestream/mainstream ratios quoted in Table 1 can be misleading if used out of context. The important question is not the ratio of sidestream/mainstream but rather what is the concentration of the constituent in the indoor environment and how does it compare to levels from sources other than ETS. Studies based solely on observations of fresh sidestream, or highly and unrealistically concentrated ETS, should take into account the possible differences between these smokes and ETS found in real-life situations.

2023380075

- Even the 1986 Report of the Surgeon General on ETS and the 1986 NRC/NAS Report on ETS conceded:

Comparison of the relative concentrations of various components of SS and MS smoke provides limited insights concerning the toxicological potential of ETS in comparison with active smoking. As described above, SS characteristics, as measured in a chamber, do not represent those of ETS, as inhaled by the non-smoker under nonexperimental conditions.<sup>2</sup>

Similarly, the NAS Report concluded:

Because the physicochemical nature of ETS, MS, and SS differ, the extrapolation of health effects from studies of MS or of active smokers to nonsmokers exposed to ETS may not be appropriate . . .<sup>3</sup>

2023380076

#### REFERENCES

1. Baker, R. and C. Proctor, "The Origins and Properties of Environmental Tobacco Smoke," Env. Int. (16): 231-245, 1990.

See also:

Löfroth, G., et al., "Characterization of Environmental Tobacco Smoke," Env Sci Technol (23): 610-614, 1989.

Scherer, G., et al., "Importance of Exposure to Gaseous and Particulate Phase Components of Tobacco Smoke in Active and Passive Smokers," Occup. Env. Health (62): 459-466, 1990.

Nystrom, C., et al., "Assessing the Impact of Environmental Tobacco Smoke on Indoor Air Quality: Current Status," Proceedings of the ASHRAE Conference, IAO '86, April 20-23, 1986, Atlanta, Georgia: 213-233, 1986.

Rawbone, R., "The Aging of Sidestream Tobacco Smoke Components in Ambient Environments," Indoor Air Quality, ed. H. Kasuga, Springer-Verlag, Berlin Heidelberg: 55-61, 1990.

Proctor, C. and H. Dymond, "The Measurement of ETS Through Adsorption/Desorption Procedures," Indoor Air Quality, ed. H. Kasuga, Springer-Verlag, Berlin Heidelberg: 82-89, 1990.

Piade, J., et al., "Assessment of ETS Impact on Office Air Quality," Indoor Air Quality, ed. H. Kasuga, Springer-Verlag, Berlin Heidelberg: 112-119, 1990.

2. U.S. Department of Health and Human Services, A Report of the Surgeon General, The Health Consequences of Involuntary Smoking: 23-25, 1986.
3. Committee on Passive Smoking, Board on Environmental Studies and Toxicology, National Research Council, Environmental Tobacco Smoke, Measuring Exposures and Assessing Health Effects, National Academy Press, Washington, D.C.: 7-9, 1986.

2023380077

2

2023380078

## EXPOSURE TO ETS

- Published studies indicate that nonsmoker exposure to ETS under normal, everyday conditions is minimal. For example, researchers report that there is little difference in ambient levels of carbon monoxide in smoking and nonsmoking areas of workplaces and public places and in homes with and without smokers.<sup>1-6</sup> Other studies indicate that ETS contributes less than half of the total particles in the air of a typical public place.\*<sup>7-14</sup> Nicotine is often used as a marker for ETS
- \* A paper published in a 1980 issue of Science magazine, in which the authors reported the results of their efforts to measure particles or particulates in the air of smoking and nonsmoking areas, is often cited to support the claim that ETS is a major indoor pollutant. The authors, Repace and Lowrey, contend that the levels of particles they observed in the smoking areas were much higher than in the nonsmoking areas. However, their study results are inconsistent with many others. For example, the average particle count attributed to ETS in their study was from three to twenty times higher than the average levels reported in other studies of office buildings, restaurants and residences.

There are a number of explanations for the authors' apparent overestimation of ETS exposure. First, they selectively sampled environments such as meeting and game rooms, bars and sandwich shops which did not represent normal occupancy conditions and where particulate levels would likely be high regardless of the presence or absence of tobacco smoke. Second, through inappropriate testing methods, they incorrectly assumed all particles in the air arose from ETS. However, as several researchers have noted, ETS typically contributes about one-third of the overall particle levels in indoor spaces. Moreover, particles also are generated by people and their everyday routine activities such as movement and cooking. (Repace, J. and A. Lowrey, "Indoor Air Pollution, Tobacco Smoke and Public Health," Science 208: 464-472, 1980.)

20233800029

exposures because it is unique to tobacco smoke. Typical measurements of nicotine range from an exposure equivalent of 1/100 to 1/1000 of one filter cigarette per hour.<sup>15-22</sup> This means that a nonsmoker would have to spend from 100 to 1000 hours in an office, restaurant or public place in order to be exposed to the nicotine equivalent of a single cigarette.

- Studies which have examined ETS constituent levels of nitrosamines, nitrogen oxides and volatile organic compounds (such as benzene\*\*) report minimal contributions to overall ambient air levels in homes, the workplace and public places.<sup>23-36</sup>

---

\*\* Benzene exposure from ETS is negligible, despite reports to the contrary.<sup>37-38</sup> "Automotive fuel is, by far, the largest, most pervasive source of benzene exposure. In 1989, the U.S. Department of Health and Human Services estimated that 1 billion pounds of benzene were released into the atmosphere from the refueling and operation of approximately 130 million motor vehicles in 1976 [NIEHS, 1989]. This translates into 7.8 pounds of benzene per vehicle per year. In contrast, a pack-per-day smoker would generate approximately 0.008 pounds of benzene per year, assuming that, at most, 0.5 mg of benzene is generated from one cigarette (MS plus SS) [Hoffmann, 1990]. Based on these estimates, an average person is potentially exposed to 1,000 times more ambient benzene from one automobile than from a smoker in a given year." [From: Response of RJR, The U.S. EPA: "ETS: A Guide to Workplace Smoking Policies," October 1, 1990.]

2023380080

Questionnaire Reliability:

- All of the epidemiologic studies on the purported association between ETS exposure and disease in nonsmokers rely solely upon questionnaires about exposure, rather than upon actual exposure data.<sup>39-41</sup> Recent studies indicate that questionnaires are an unreliable and inaccurate measure of exposure. Questionnaire responses about exposure vary widely when compared with actual measurements of ETS constituents in the ambient air.<sup>41</sup>

ETS and Radon:

- A theory that suggests that concentrations of radon decay products increase in the presence of tobacco smoke, thus implying an increased risk of lung cancer for the nonsmoker, has been reported in the literature.<sup>42-44</sup> The theory suggests that radon decay products attach to particles (including ETS) in the air, remain suspended, and are subsequently taken up in the lungs of nonsmokers.
- However, actual data indicate that this is not the case.<sup>45-48</sup> It is the unattached, gaseous fraction of radon which determines the dose of radiation to the respiratory tract. According to these data, as dust or particulate levels

2023380081

increase, the unattached fraction of radon daughters will decrease, thereby lowering the potential dose of radiation to the lungs.

2023380082

## REFERENCES

1. Kirk, P., et al., "Environmental Tobacco Smoke in Indoor Air," Indoor and Ambient Air Quality, eds. R. Perry and P. Kirk, London, Selper Ltd.: 99-112, 1988.
2. Duncan, D. and P. Greavey, "Passive Smoking and Uptake of Carbon Monoxide in Flight Attendants," JAMA 251(20): 120-21, 1984.
3. Cox, B. and M. Whichelow, "Carbon Monoxide Levels in the Breath of Smokers and Nonsmokers: Effect of Domestic Heating Systems," J Epidemiol Community Health 39: 75-78, 1985.
4. Girman, J. and G. Traynor, "Indoor Concentrations," J Am Pollut Control Assoc 33(2): 89, 1983.
5. Yocom, J., "Indoor Concentrations," JAPCA, 33(2): 89, 1983.
6. Nitta, H., et al., "Measurements of Indoor Carbon Monoxide Levels Using Passive Samplers in Korea," Indoor Air '90: The Fifth International Conference on Indoor Air Quality and Climate, Toronto, Canada, July 29-August 3: 77-82, 1990.
7. Carson, J. and C. Erikson, "Results from Survey of Environmental Tobacco Smoke in Offices in Ottawa, Ontario," Environ Technol Letters 9: 501-508, 1988.
8. Sterling, T., et al., "Environmental Tobacco Smoke and Indoor Air Quality in Modern Office Work Environments," J. Occup. Med. 26(1): 57-62, 1987.
9. Sterling T. and B. Mueller, "Concentrations of Nicotine, RSP, CO and CO<sub>2</sub> in Nonsmoking Areas of Offices Ventilated by Air Recirculated From Smoking Designated Areas," Am. Ind. Hyg. Assoc. J., 49(9): 423-426, 1988.
10. Hosein, R., "The Relationship Between Pollutant Levels in Homes and Potential Sources;" Indoor Air Quality in Cold Climates, Hazards and Abatement Measures, ed. D. Walkinshaw, Ottawa, Ontario, APCA: 250-260, 1985.
11. Proctor, C., et al., "Measurements of Environmental Tobacco Smoke in an Air-Conditioned Office Building," Environ. Technol. Letters (10): 1003-1018, 1989.

2023380083

- 2023380084
12. Oldaker, G., et al., "Results From Surveys of Environmental Tobacco Smoke in Restaurants," Indoor Air Quality, ed. H. Kasuga, Springer-Verlag, Berlin Heidelberg: 99-104, 1990.
  13. Spengler, J., "Harvard's Indoor Air Pollution Health Study," Indoor Air Quality, H. Kasuga (ed.), Springer-Verlag, Berlin Heidelberg: 241-248, 1990.
  14. Quackenboss, J. and Lebowitz, M., "Indoor-Outdoor Relationships for Particulate Matter: Exposure Classifications and Health Effects," Environ. Int., 15: 353-360, 1989.
  15. Hinds, W. and First, M., "Concentrations of Nicotine and Tobacco Smoke in Public Places," New Eng. J. Med. 292(16): 844-845, 1975.
  16. Badre, R., et al., "Pollution Atmospherique par la Fumee de Tabac (Atmospheric Pollution by Smoking)," Ann. Pharm. Fr. 36(9-10): 443-452, 1978. Translation.
  17. Jenkins, R., et al., "Development and Application of a Thermal Desorption-Based Method for the Determination of Nicotine in Indoor Environments," Indoor and Ambient Air Quality, eds. R. Perry and P. Kirk, London, Selper Ltd.: 557-566, 1988.
  18. Muramatsu, M., et al., "Estimation of Personal Exposure to Tobacco Smoke with a Newly Developed Nicotine Personal Monitor," Environ. Res. 35: 218-227, 1984.
  19. Muramatsu, M., et al., "Estimation of Personal Exposure to Ambient Nicotine in Daily Environment," Arch. Occup. Environ. Health 59: 545-550, 1987.
  20. Thompson, C., et al., "A Thermal Desorption Method for the Determination of Nicotine in Indoor Environments," Envir. Sci. Tech. 23: 429-435, 1989.
  21. Foliart, D., et al., "Passive Absorption of Nicotine in Airline Flight Attendants," New Eng. J. Med., 308(18): 1105, 1983.
  22. Oldaker, G. and Conrad, F., "Estimation of the Effect of Environmental Tobacco Smoke on Air Quality Within Passenger Cabins of Commercial Aircraft," Envir. Sci. Tech. 21: 994-999, 1987.
  23. Lehnert, G., "Conclusion: In the Realm of Speculation," Munch med Wschr, 124 (1982) Nr. 4.

24. Schievelbein, H., "Are There Really Important New Findings About Passive Smoking?", Oeff Gesundheitswes, 44(0): 454-56, 1982. Translation.
25. Stehlik, G., et al., "Concentration of Dimethylnitrosamine in the Air of Smoke-Filled Rooms," Ecotoxicol Environ Safety 6: 495-500, 1982.
26. Sega, K. and M. Fugas, "Nitrogen Dioxide Concentrations in Residences," Indoor and Ambient Air Quality, eds. R. Perry and P. Kirk, London, Selper Ltd.: 493-496, 1988.
27. Good, B., et al., "Effect of Cigarette Smoking on Residential NO<sub>2</sub> Levels," Environ Int 8: 167-175, 1982.
28. Shimizu, Y., et al., "Influence of Indoor Air Pollution on NO<sub>2</sub> Personal Exposure Levels of Schoolchildren," Present and Future of Indoor Air Quality, eds. C. Bieva, et al., Brussels, Elsevier: 337-345, 1989.
29. Hendel-Kramer, A., et al., "Regression Model for Indoor Concentrations of Combustion-Generated Gases," Present and Future of Indoor Air Quality, eds. C. Bieva, et al., Brussels, Elsevier: 315-320, 1989.
30. Bayer, C. and Black, M., "Thermal Desorption/Gas Chromatographic/Mass spectrometric Analysis of Volatile Organic Compounds in the Offices of Smokers and Nonsmokers," Biomed and Envir Mass Spect 14(8): 363-367, 1987.
31. Proctor, C., et al., "Measurements of Environmental Tobacco Smoke in an Air-Conditioned Office Building," Environ. Technol. Letters (10): 1003-1018, 1989.
32. Godish, T., "Formaldehyde Exposures from Tobacco Smoke: A Review," AJPH 79(8): 1044-1045, 1989.
33. Godish, T., "Residential Formaldehyde: Increased Exposure Levels Aggravate Adverse Health Effects," J. of Environ. Health 53(3): 34-35, 1990.
34. Adlkofer, F., et al., "Significance of Exposure to Benzene and Other Toxic Compounds Through Environmental Tobacco Smoke," J. Cancer Res. Clin. Oncol. (116): 591-598, 1990.
35. Hugod, C., et al., "Exposure of Passive Smokers to Tobacco Smoke Constituents," Int. Arch. Occup. Environ. Health (42): 21-29, 1978.

2023380085

36. Proctor, C., et al., "Measurement of Environmental Tobacco Smoke in an Air-Conditioned Office Building," Present and Future of Indoor Air Quality, eds. C.J. Bieva, et al., Brussels, Elsevier: 169-172, 1989.
37. Hattemer-Frey, H., "Benzene: Environmental Partitioning and Human Exposure," Environ. Res., 53: 221-232, 1990.
38. Wallace, L., "Major Sources of Exposure to Benzene and Other Volatile Organic Chemicals," Risk Analysis, 10(1): 59-64, 1990.
39. Coultas, D., et al., "Questionnaire Assessment of Lifetime and Recent Exposure to Environmental Tobacco Smoke," Am J of Epidemiol. 130(2): 338-347, 1989.
40. Coultas, D., et al., "Variability of Measures of Exposure to Environmental Tobacco Smoke in the Home," Am Rev Resp Dis 142: 602-606, 1990.
41. Coultas, D., et al., "A Personal Monitoring Study to Assess Workplace Exposure to Environmental Tobacco Smoke," American J. of Public Health 80(8): 988-990, 1990.
42. Bergman, H. and Axelson, O., "Passive Smoking and Indoor Radon Daughter Concentrations," Lancet: 1308-09, 1983.
43. Bergman, H., et al., "Indoor Radon Daughter Concentrations and Passive Smoking," Env. Int. (12): 17-19, 1986.
44. Moghissi, A. and M. Seiler, "Enhancement of Exposure to Radon Progeny as a Consequence of Passive Smoking," Env. Int. (15): 261-64, 1989.
45. Upfal, M., et al., "Indoor Radon and Lung Cancer in China," J. Nat. Cancer Inst.: 1722, 1989.
46. Stebbings, J. and J. Dignam, "Contamination of Individuals by Radon Daughters: A Preliminary Study," Archives of Environ. Health 43(2): 149-54, 1988.
47. Pritchard, J. and J. Strong, "The Influence of Environmental Tobacco Smoke on Radon Dosimetry," Indoor Air '90: The Fifth International Conference on Indoor Air Quality and Climate, Toronto, Canada, July/August 1990: 33-38.
48. McAughay, J., et al., "Risk Assessment of Exposure to Indoor Air Pollutants," Env. Int. (11): 295-302, 1990.

2023380086

**2023380087**

#### DETERMINATION OF DOSE: COTININE

- It has been reported that cotinine, a substance converted from nicotine by the body, can be used as a biological marker to measure nonsmoker exposure to ETS.<sup>1-2</sup> While some reports suggest that cotinine is a reliable marker for total exposure to tobacco smoke, many others do not.<sup>3-12</sup> Researchers have reported that individuals metabolize nicotine in different ways at different times and that elimination rates for cotinine vary among individuals. In addition, recent research indicates that diet may contribute to levels of nicotine and cotinine found in the body, thereby interfering with ambient air exposure levels.<sup>13</sup> Scientists have also noted that different methods of analysis may influence final recorded levels of cotinine.<sup>14</sup> And finally, because cotinine is a metabolite of a gas-phase constituent of ETS, nicotine, cotinine levels do not represent exposures to other constituents of ETS.
- In conclusion, cotinine is not a reliable quantitative measure of ETS exposure. This is because body fluid levels of cotinine cannot be attributed solely to nicotine in ETS, and because body fluid levels of cotinine do not correlate well with actual ambient air exposures to ETS or with ETS constituents other than nicotine. At best, cotinine may be used as a qualitative marker of ambient nicotine exposures.

2023380088

REFERENCES

1. Wald, N., et al., "Urinary Cotinine as Marker of Breathing Other People's Tobacco Smoke," Lancet I, 230-231, 1984.
2. Watts, R. et al., "Cotinine Analytical Workshop Report: Consideration of Analytical Methods for Determining Cotinine In Human Body Fluids as a Measure of Passive Exposure to Tobacco Smoke," Environ. Health Pers. 84: 173-182, 1990.
3. Curvall, M., et al., "Simulation and Evaluation of Nicotine Intake During Passive Smoking: Cotinine Measurements in Body Fluids of Nonsmokers Given Intravenous Infusions of Nicotine," Clin. Pharmacol. Ther. 47(1): 42-49, 1990.
4. Itani, S., et al., "A Comparison of Plasma and Urinary Nicotine and Cotinine Levels in Smokers and Nonsmokers: Nicotine Excretion Pathways are Possibly Differential According to the Dosage of Tobacco Smoke Uptake," Indoor Air Quality, ed. H. Kasuga, Springer-Verlag, Berlin Heidelberg: 202-212, 1990.
5. Idle, J., "Commentary: Titrating Exposure to Tobacco Smoke Using Cotinine -- A Minefield of Misunderstanding," J. Clin. Epidemiol. 43(4): 313-317, 1990.
6. Biber, A., et al., "Determination of Nicotine and Cotinine in Human Serum and Urine: An Interlaboratory Study," Toxicology Letters 35(1): 45-52, 1987.
7. Haley, N. et al., "Elimination of Cotinine from Body Fluids: Disposition in Smokers and Nonsmokers," AJPH 79(8): 1046-1048, 1989.
8. Cummings, K., et al., "Measurement of Current Exposure to Environmental Tobacco Smoke," Archives of Environ. Health 45(2): 74-79, 1990.
9. Lee, P., "Lung Cancer and Passive Smoking," Br. J. Cancer, 63: 161-162, 1991.
10. Johnson, L., et al., "Passive Smoking Under Controlled Conditions," Int. Arch. Occup. Environ. Health 56: 99-110, 1985.
11. Letzel, H., et al., "Measuring Problems in Estimating the Exposure to Passive Smoking Using the Excretion of Cotinine," Toxicology Letters 35(1): 35-44, 1987.

2023380089

12. Schwartz, S., et al., "Mathematical Modelling of Nicotine and Cotinine as Biological Markers of Environmental Tobacco Smoke Exposure," Toxicology Letters 35(1): 53-58, 1987.
13. Castro, A. and N. Monji, "Dietary Nicotine and Its Significance in Studies on Tobacco Smoking," Bio Arch 2(2): 9197, 1986.
14. Adlkofer, F., et al., "Passive Smoking," N Engl J Med 312(11): 719-720, 1984.

2023380090

DETERMINATION OF DOSE: LUNG RETENTION

- Cotinine is a biologically inactive substance which has not been correlated with ETS constituents retained in the lung. Several researchers have estimated levels of ETS particulate uptake by nonsmokers to approximate 0.02% (two-hundredth of one percent) that of the particulate exposure of an active smoker.<sup>1-4</sup>

2023380091

#### REFERENCES

1. Arundel, A., et al., "Never Smoker Lung Cancer Risks from Exposure to Particulate Tobacco Smoke," Environ Int 13: 409-426, 1987.
2. Adlkofer, F., "Biological Effects After Exposure to ETS," Indoor Air Quality: Symposium, Buenos Aires, National Academy of Sciences of Buenos Aires: 61-76, 1989.
3. McAughey, J., et al., "Respiratory Deposition of Environmental Tobacco Smoke," Indoor Air '90: The Fifth International Conference on Indoor Air Quality and Climate, Toronto, Canada, July/August: 361-366, 1990.
4. Comments of the R.J. Reynolds Tobacco Company on Appendix C to the EPA Draft Risk Assessment - Dosimetry of Environmental Tobacco Smoke, September 1990, B-1 - C-7.

2023380092

### DETERMINATION OF DOSE: MUTAGENS

- Some reports have suggested that the potential toxicity of ETS can be assessed by measuring mutagens in the body fluids of nonsmokers exposed to ETS.<sup>1-3</sup> Mutagens are substances capable of altering the genetic structure of cells. It is suggested that the presence of mutagens in body fluids (e.g. urine) may be an indication that an individual has been exposed to substances capable of inducing cancer.
- Impetus for the theory arises, in part, from studies which report that various constituents of ETS collected through airborne samples are capable of inducing mutations in bacteria.<sup>4-6</sup>
- However, the significance of such reported findings has not been established. Virtually all air samples, whether in the presence or absence of smoking, are mutagenic. Indeed, no substance, including food and natural materials, has been unequivocally shown to be free of carcinogenic and/or mutagenic properties. In addition, it has been reported that sidestream smoke exhibits diminished mutagenic activity as it ages and becomes diluted (i.e., as it becomes ETS).<sup>7</sup>

2023380093

- With few exceptions, studies which have compared mutagens in the body fluids of nonsmokers exposed to realistic levels of ETS and nonsmokers not exposed to ETS report no significant difference in mutagenic activity.<sup>8-11</sup>
- The few studies reporting significant increases in urinary mutagenicity among individuals exposed to ETS<sup>1-3</sup> did not employ realistic levels of exposure to ETS, and they did not control adequately for the presence of mutagens in the diet of the study subjects.

2023380094

REFERENCES

1. Bos, R., et al., "Excretion of Mutagens in Human Urine After Passive Smoking," Cancer Letters (19): 85-90, 1983.
2. Husgafvel-Pursiainen, K., et al., "Passive Smoking at Work: Biochemical and Biological Measures of Exposure to Environmental Tobacco Smoke," Int. Arch. Occup. Environ. Health (59): 337-345, 1987.
3. Mohtashamipur, E., et al., "Urinary Excretion of Mutagens in Passive Smokers," Toxicol. Letters (35): 141-146, 1987.
4. Claxton, L., et al., "A Genotoxic Assessment of Environmental Tobacco Smoke Using Bacterial Bioassays," Mutation Res. (222): 81-99, 1989.
5. Lofroth, G., et al., "Public Exposure to Environmental Tobacco Smoke," Mutation Res. (202): 103-110, 1988.
6. Matsushita, H., et al., "Highly Sensitive Methods for the Evaluation of Carcinogens and Mutagens Indoor," Indoor Air Quality, ed. H. Kasuga, Springer-Verlag, Berlin Heidelberg: 371-382, 1990.
7. Sonnenfeld, G. and D. Wilson, "The Effect of Smoke Age and Dilution on the Cytotoxicity of Sidestream (Passive) Smoke," Toxicol. Letters (35): 89-94, 1987.
8. Scherer, G., et al., "Urinary Mutagenicity After Controlled Exposure to Environmental Tobacco Smoke (ETS)," Toxicology Letters 35(1): 135-140, 1987.
9. Bombick, D., et al., "Assessment of the Biological Activity of Mainstream or Environmental Tobacco Smoke (ETS) Using a Cellular Smoke Exposure Technique," Abstracts of the Twenty-Second Annual Scientific Meeting of the Environmental Mutagen Society, Kissimmee, Florida: April 1991.
10. Scherer, G., "Biomonitoring of Exposure to Potentially Genotoxic Substances from Environmental Tobacco Smoke," Environ. Int. (15): 49-56, 1989.
11. Scherer, G., "Quantitative and Qualitative Differences in Tobacco Smoke Uptake Between Active and Passive Smoking," Indoor and Ambient Air Quality, eds. R. Perry and P. Kirk, Selper Ltd., London: 189-194, 1988.

2023380095

DOSE: OTHER BIOLOGICAL MARKERS

- It has been suggested that sidestream smoke (and by inference, ETS) contains polycyclic aromatic hydrocarbons (PAH), substances which have been designated as carcinogens by various governmental agencies. However, in a series of papers, German researchers report no significant differences in urinary PAH by-products among nonsmokers exposed to ETS and those not exposed.<sup>1-3</sup> Diet was reported to have a profound influence on PAH by-product formation in all study subjects.
- Japanese scientists have reported that individuals exposed to ETS have increased urinary levels of hydroxyproline (HOP), a substance believed to act as a marker for the breakdown of lung tissue.<sup>4</sup> However, German researchers have reported no increase in HOP excretion among either smokers or nonsmokers exposed to ETS.<sup>5</sup>
- It has recently been suggested that DNA adducts can be utilized as biomarkers to assess exposure (dose) to ETS.<sup>6</sup> (An adduct is a product derived from reactions between chemicals and biological material (such as DNA)). Research, however, does not conclusively support this theory; nonsmokers exposed to ETS do not appear to exhibit increased DNA adduct production.<sup>7</sup> Other studies report no increased chromosomal changes in body

2023380096

fluids of nonsmokers exposed to ETS.<sup>8-9</sup>

2023380097

### REFERENCES

1. Scherer, G., et al., "Urinary Mutagenicity, Hydroxy-Phenanthrene, and Thioether Excretion After Exposure to Environmental Tobacco Smoke," Indoor Air Quality, ed. H. Kasuga, Springer-Verlag, Berlin Heidelberg: 138-146, 1990.
2. Martin, F., et al., "Urinary Excretion of Hydroxy-Phenanthrenes After Intake of Polycyclic Aromatic Hydrocarbons," Environ. Int. (15): 41-47, 1989.
3. Hoepfner, H., et al., "Hydroxy-Phenanthrenes in the Urine of Non-Smokers and Smokers," Toxicology Letters (35): 67-71, 1987.
4. Kasuga, H., "An Introduction to the Study of Smoking Using Urinary Hydroxyproline," Indoor Air Quality, ed. H. Kasuga, Springer-Verlag, Berlin Heidelberg: 37-52, 1990.
5. Adlkofer, F., "The Significance of Urinary Hydroxyproline Excretion in Smokers and Passive Smokers," Indoor Air Quality, ed. H. Kasuga, Springer-Verlag, Berlin Heidelberg: 213-218, 1990.
6. Perera, F., et al., "DNA Adducts, Protein Adducts, and Sister Chromatid Exchange in Cigarette Smokers and Nonsmokers," JNCI 79(3): 449-456, 1987.
7. Holz, O., et al., "<sup>32</sup>P-postlabelling Analysis of DNA Adducts in Monocytes of Smokers and Passive Smokers," Int. Arch. Occup. Environ. Health, 62: 299-303, 1990.
8. Sorsa, M., et al., "Cytogenetic Effects of Tobacco Smoke Exposure Among Involuntary Smokers," Mutation Res. 222(2): 111-116, 1989.
9. Husgafvel-Pursiainen, K., "Sister-Chromatid Exchange and Cell Proliferation in Cultured Lymphocytes of Passively and Actively Smoking Restaurant Personnel," Mutation Res. 190: 211-215, 1987.

2023380098

4

2023380099

### BIOLOGICAL PLAUSIBILITY

- The argument for the biological plausibility of the role of ETS in disease causation depends upon the simplistic claim that since mainstream (MS) and sidestream (SS) smoke contain carcinogenic substances, so must ETS. However, this analogy is not proved.
- ETS has never been shown to be carcinogenic in any animal species. Only two animal inhalation experiments investigating ETS and lung cancer have been published. Both studies report no meaningful histopathological differences between animals exposed to ETS and those which were not exposed. In a study conducted by the American Health Foundation,<sup>1-3</sup> the investigators exposed one group of hamsters to mainstream smoke and another group to ETS. Animals exposed to mainstream smoke and ETS lived longer than the sham treated controls. The investigators reported that overall there was no marked increase in tumor incidence in animals exposed to either mainstream smoke or ETS after 18 months of exposure. The second study was a 90-day ETS inhalation study of rats and hamsters.<sup>4</sup> Animals were exposed to ETS concentrations 100 times those concentrations encountered by nonsmokers. These researchers reported no histopathological differences between exposed and control animals. Electron microscopy revealed

2023380100

pulmonary changes which could be expected to occur under similar exposure conditions with other substances.

- In addition, recent reviews of the literature on suspected pulmonary carcinogens have indicated that none of the individual constituents in sidestream smoke classified as potentially carcinogenic has been found to induce pulmonary cancer via inhalation in experimental animals.<sup>5-6</sup>
- ETS has not be shown to be mutagenic in any animal or cell culture system when tested at realistic levels of exposure (See Section III).
- These points undermine the credibility of the argument for the biological plausibility of ETS in disease causation.

2023380101

## REFERENCES

1. Haley, N., et al., "Uptake of Sidestream Smoke by Syrian Golden Hamsters," Toxicol. Letters 35 (1987): 83-88.
2. Haley, N., "Sidestream Smoke Uptake By Syrian Golden Hamsters in An Inhalation Bioassay," Indoor Air '87: Proceedings of the 4th International Conference on Indoor Air Quality and Climate, Institute for Water, Soil and Air Hygiene, Berlin: 68-73, 1987.
3. American Health Foundation, "Experimental Tobacco Carcinogenesis," Application for Continuation Grant to Department of Health and Human Services, U.S. Public Health Service, May 1988.
4. Adlkofer, F., et al., "Exposure of Hamsters and Rats to Sidestream Smoke of Cigarettes: Preliminary Results of a 90-Day-Inhalation Study," Indoor and Ambient Air Quality, eds. R. Perry and P. Kirk, London, Selper Ltd.: 252-258, 1988.
5. Aviado, D., "Suspected Pulmonary Carcinogens in Environmental Tobacco Smoke," Environ. Tech. Letters, 9: 539-544, 1988.
6. Aviado, D., "Health Effects of 50 Selected Constituents of Environmental Tobacco Smoke," Indoor Air Quality, ed. H. Kasuga, Springer-Verlag, Berlin Heidelberg: 383-389, 1990.

2023380102

5

2023380103

**2023380104**

**APPENDIX I**

## ASSESSING EXPOSURE TO ENVIRONMENTAL TOBACCO SMOKE: IS IT VALID TO EXTRAPOLATE FROM ACTIVE SMOKING?

M.J. Reasor, J.A. Will<sup>1</sup>

Department of Pharmacology & Toxicology, West Virginia University Health Sciences Center, Morgantown, WV, USA and <sup>1</sup>Department of Veterinary Science, University of Wisconsin-Madison, Madison, WI, USA

### Abstract

This review examines the question of whether exposure to environmental tobacco smoke (ETS) can be assessed by extrapolation from active smoking. General problems associated with assessing exposure to ETS and the pathophysiological consequences are discussed. Among the topics presented are the dynamic chemical and physical characteristics of ETS and exposure assessment using airborne and biological markers. The reported pathophysiological consequences of ETS exposure are examined in the context of dose and exposure. The conclusion is that it is extremely difficult, if not impossible, to extrapolate from active smoking to ETS exposure with any degree of reliability.

**Key words:** Environmental tobacco smoke, nicotine, cotinine, adducts, cancer, risk assessment, pathophysiology

### Introduction

Tobacco smoke is an exceedingly complex matrix, consisting of several thousand constituents. As it is dispersed in the atmosphere, its chemical and physical complexity can be increased through reactions among its constituents and through evaporation, condensation, coagulation and adsorption or impaction on surfaces [1]. Tobacco smoke as it exists in the ambient environment is termed environmental tobacco smoke (ETS) and is clearly a complex and dynamic material whose properties are influenced by numerous factors. With recent concern that exposure to ETS may present a health hazard to the non-smoker [2,3], a number of risk assessments have been published

dealing principally with the possible relationship of ETS to mortality and lung cancer [4]. Among several approaches used for ETS risk assessment has been the comparison between exposure to ETS and active smoking [5-8]. Inherent in such an approach is the assumption that ETS is a dilute form of mainstream smoke (MS) inhaled during active smoking, and that other than the differences in concentration, exposure conditions are similar. Considerable information exists concerning the properties of MS and the conditions of exposure during active smoking [9,10], in large part because material can be collected under reproducible conditions that simulate those to which the smoker is exposed. In contrast, the

*Address correspondence to Dr M.J. Reasor, Department of Pharmacology & Toxicology, West Virginia University Health Sciences Center, Morgantown, WV 26506, USA.*

2023380105

dynamic nature of ETS precludes its characterisation and assessment of exposure to a degree of accuracy possible with mainstream smoke.

Certain criteria must be considered in conducting a risk assessment of a material [11]. Three of these involve consideration of composition and exposure:

1. *A hazard identification*, in which it is determined whether a particular substance is causally linked to specific health effects.
2. *An exposure assessment*, in which the extent humans are exposed to the material has been determined.
3. *A dose-response assessment*, in which the amount of exposure to the material and the probability of occurrence of the specific health effects have been determined. It is the purpose of this article to examine the question of whether exposure to ETS can be assessed by extrapolation from active smoking.

### Characterisation of ETS

The following discussion will be concerned with cigarettes only. Mainstream smoke (MS) is that smoke drawn into the mouth through the butt end of the cigarette by the active smoker. Side-stream smoke (SS) is defined as all other smoke emitted from the cigarette with the vast majority being the smoke released from the burning end of the cigarette between puffs [12]. In addition to MS, the active smoker is exposed to SS at levels higher than the non-smoker because of the proximity to its generation. ETS is composed of both SS and exhaled mainstream smoke (EMS), the material exhaled by the active smoker. While it is generally accepted that SS makes a larger contribution to ETS than does EMS, the relative contributions of each material to ETS have not been systematically examined. There is some evidence that EMS contributes little to the gas phase of ETS, however, it does contribute significantly to the particulate phase of ETS [12]. With certain tobaccos,

EMS may contribute over 40% of the particles of ETS.

The properties of ETS are influenced significantly by a number of considerations including type of tobacco smoked and smoke density, as well as environmental factors such as dilution, ventilation, temperature, humidity, lighting and adsorption onto surfaces. Additionally, chemical reactions occur changing the composition of ETS; e.g. with time after generation, nitric oxide is converted to nitrogen dioxide [12]. The changes that occur as ETS lingers indoors are termed aging, and contribute significantly to the complexity and dynamic nature of ETS. Because of these factors, it is impossible to provide a definitive chemical and physical description of ETS, its character differing depending on conditions that exist at any given time. As a result, little consistent information exists on the characteristics of ETS under ambient conditions in indoor environments that would allow generalisations about its composition to be made. Because the frequency of puffing and the depth of inhalation differ among smokers, it should be apparent that the relative contributions of SS and EMS to ETS will be different for each ambient environment. Therefore, in addition to environmental factors described previously, the chemical and physical properties of ETS are dependent upon the smoking patterns that occur in an indoor environment. The origins and properties of ETS have been reviewed in detail elsewhere [12].

These considerations notwithstanding, numerous studies have been conducted in an attempt to characterise ETS. These have included the analysis of freshly generated SS, SS allowed to age in controlled environmental chambers, SS allowed to age in well-controlled experimental indoor environments, and ETS in a number of typical indoor environments. Each of these situations has specific limitations as to its usefulness in characterising ETS.

Considerable effort has been directed at characterising freshly generated SS as

2023380106

a surrogate for ETS, and much data exist on the chemical composition of this material [13-19]. Serious problems are inherent in utilising this approach. First, and most importantly, ETS is much more complex and variable than SS generated in the laboratory due to the presence of undefined proportions of both SS and EMS and the influence of aging on ETS components. Secondly, SS is produced under conditions that do not necessarily represent the smoking pattern of individuals. SS is generated under standardised smoking conditions adopted over 20 years ago in apparatuses that allows it to be rapidly collected for analysis. The conditions are almost always one puff/min of 2 sec duration and a volume of 35ml. Since people smoke with different patterns these conditions do not necessarily simulate those of most smokers [20], and as a result, the quantities of materials released into ambient air will likely vary from those generated using smoking machines. The same objections about standardised smoking conditions could be raised regarding the composition of MS, as well.

The environmental conditions present during generation will influence the levels of chemicals in SS. This is illustrated by the effect of the velocity of air passed over the tip of the burning cigarette when generating SS [21]. In this study, the level of dimethylnitrosamine in SS varied as a function of air flow. Flow rates of 250, 500, 1000, and 1500ml of air/min yielded levels of dimethylnitrosamine of 90, 250, 530, and 680ng/cigarette, respectively. Therefore, depending on the conditions used for generation and collection, values for SS may vary greatly. This is illustrated by the wide range of values reported in the literature for nearly one hundred chemicals reported to be present in SS [15].

Compared to the study of freshly generated SS, utilisation of environmental chambers offers the opportunity to examine the properties under controlled, although not necessarily realistic conditions. The most extensive examination

of SS-derived ETS under these conditions appears to have been performed by Eatough and colleagues [14,15,22]. They have utilised an unventilated teflon chamber in studying the properties of ETS originating from SS generated within the chamber. Use of the teflon chamber permits ETS to be studied in a setting where results are not influenced by ventilation or surface properties. Under these conditions, a comprehensive analysis of the chemical composition of the gas and particle phases of ETS was performed, and the behaviour of the particulate phase examined. For example, it was observed that nitrogen dioxide was the major inorganic acid present in the gas phase of ETS, and nicotine, 3-ethenylpyridine, and pyridine were the principal nitrogen bases present. Major particulate phase organic compounds were nicotine, myosmine, solanesol, nicotyrine and cotinine. Greater than 95% of the nicotine was present in the gas phase. As the ETS aged, particles underwent at least three changes. Particles deposited on the wall of the chamber, they coagulated increasing in size, and evaporation from the particles was also significant. The effect of UV radiation was also examined, and it was noted that the level of gas phase nicotine decreased with a concomitant, but less than stoichiometric increase in particulate phase nicotine. An important class of compounds, the nitrosamines were not examined in this system. It will be of considerable interest when the levels and behaviour of the volatile and tobacco-specific nitrosamines are examined under such controlled conditions.

Using a stirred stainless steel chamber to study the properties of ETS, it was reported that smoke particles underwent evaporation over the first few hours [23]. As the ETS aged, particle size increased due to a combination of coagulation and removal of smaller particles by deposition on the surface of the chamber. Similar observations have been made using a ventilated steel chamber [24].

The decay of a number of SS-derived components has also been studied in a

2023380102

non-ventilated glass and stainless steel chamber [25]. In particular, polycyclic aromatic hydrocarbons (PAHs) decay at different rates during aging depending on their molecular weights; PAHs below 156 daltons had a longer half-life than those above this value. As with other experimental systems, nicotine decayed more rapidly than particulate material.

Studies have been reported using a modified trailer in which conditions can be controlled with respect to ventilation, temperature, humidity, and circulation [15,22]. Such an environment can be made to simulate closely ambient indoor conditions. Some important observations were made concerning the behaviour of SS-derived ETS in this setting which were similar to those of other workers [12]. For example, the absolute decay of various constituents of ETS was primarily controlled by the rate of ventilation. The rate of decay of nicotine was the most rapid of the components studied, while the NOx-NO species were the most stable.

While controlled chamber studies have provided useful information about ETS, the results must be interpreted with a degree of caution. These conditions only partially simulate the ambient environment in which non-smokers are exposed to ETS. For example, no studies have examined the behaviour of ETS when persons are present in the chamber or when ETS has been generated by smokers so that EMS is also present. In an effort to obtain realistic data on ETS exposure, numerous studies have examined selected chemicals and particles in ETS under a variety of ambient conditions [26-32].

Problems exist in the interpretation of these data as well. In general, only a few substances have been investigated in each study, with sampling performed over single periods of relatively short time (24 h or less). Such a sampling protocol will fail to describe the daily variations in ETS levels that exist in indoor environments as well as fail to provide a measure of chronic exposure. The lack of specificity of most of the measured substances for ETS (e.g. carbon monoxide and respirable

suspended particles) limits conclusions that can be drawn about the composition of ETS in these studies.

#### Exposure assessment methods and interpretations

It should be clear that there is no defined, reproducibly characterised entity known as ETS, rather it is a constantly changing substance influenced by numerous environmental and personal factors. At present, the published research represents little more than a broad representation of the nature of ETS. Therefore, it is currently not possible to compare the risks, if any, of exposure to ETS with those reportedly associated with active smoking based on the chemical compositions of each of these materials.

As an alternative solution to the problem of characterising ETS, several approaches have been utilised to assess ETS exposure with the goal of predicting possible related health effects. These efforts have involved the assessment of exposure by use of questionnaires, modelling, surrogate airborne markers, and the assessment of internal dose by use of biological markers (biomarkers).

Reliance on questionnaires alone to assess exposure is fraught with numerous problems including lack of standardisation and validation, responder bias and potential misclassification of subjects. At best their use represents an indirect measure of exposure and cannot provide any quantitative information on specific or total exposure levels or doses of biologically relevant chemicals at target sites [33-36]. Questionnaires can have value when used as part of a more comprehensive exposure assessment. For example, an index of exposure has been developed, which includes questionnaires as one component, along with a daily diary, that correlates well with nicotine collected by a personal monitor [37].

Modelling has been used to assess concentrations of ETS constituents and to estimate exposures [5,6,38]. Data from other studies are normally used in the modelling and, additionally, this approach

2023380108

requires assumptions which generalise and often oversimplify the exposure conditions.

The use of airborne markers and biomarkers offer the best opportunity to assess exposure to ETS. Unfortunately, reliance on either of these assessments alone for such a complex and dynamic mixture as ETS may result in misleading information. For example, the external dose may not be related to the internal dose as absorption, distribution metabolism and elimination may differ among individual components (particles, water-soluble chemicals, organic materials). The presence of a biomarker in a non-target tissue does not necessarily correlate with the level of a potentially toxic species at the critical cellular site nor whether a disease will result. These limitations in the use of airborne and biological markers are present when applied to exposure assessment for ETS.

#### *Assessment of external exposure*

Due to the complex chemical and physical nature of ETS, investigators have relied on tracers, or surrogates, in measuring external exposure to ETS. The National Research Council [2] has provided criteria which should be satisfied in using a surrogate for ETS:

1. It should be unique or nearly unique to ETS.
2. It should be present in sufficient quantity that concentrations can be easily detected in air, even at low smoking rates.
3. It should be characterised by similar emission rates for a variety of tobacco products.
4. It should be in a fairly constant ratio to the components of interest under a range of environmental conditions encountered and for a variety of tobacco products.

Unless the first criterion is fulfilled, the remaining criteria are of less significance.

To date, no single material has satisfied these criteria.

Respirable suspended particles (RSP) and nicotine have been used most frequently as surrogates for ETS. The use of RSP fails to satisfy the first criterion because of its lack of specificity to ETS. There is a significant level of background RSP not related to ETS in the indoor environment. This has been demonstrated using the property of ultraviolet absorption of RSP as representative of the ETS-specific portion of RSP [29,30]. In several environments where smoking was permitted, it was found that ETS contributed less than 40% of the particles in the indoor environment. If RSP in an indoor environment is to be attributed to ETS, it is necessary to rule out all other sources of RSP. This has not been done satisfactorily in the studies reported to date.

While the measurement of RSP may serve as an index of exposure, it is not a measure of the dose or the amount of particulate material that will be retained in the lungs of those exposed. It is the amount of material retained in the lungs that is believed to have a relationship to health effects, not the amount to which a person is exposed. In fact, the relative retention of ETS particles has never been measured.

Different deposition patterns are to be expected for the particles in ETS and those in MS because of the different breathing patterns of the two population groups. An active smoker inhales MS by mouth often with a deep inhalation followed by a prolonged respiratory pause. Such a manoeuvre increases residence time of particles and gases in the entire respiratory tract, optimising conditions for deposition. In contrast, a non-smoker would inhale ETS principally through the nose using a regular breathing pattern which is much more shallow than that used by active smokers. The shallow breathing pattern would reduce the degree of pulmonary deposition of particulate materials of ETS in non-smokers compared to MS in the active smoker.

2023380109

Risk assessments for lung cancer have been performed using estimated exposure to RSP from ETS [6,39]. The values used in these calculations were dependent on a number of assumptions that did not consider the limitations of using RSP as a surrogate for ETS. Consequently, the risk values are open to question.

Nicotine has been measured in ambient air using area sampling [40-42] and with personal samplers [37,43,44]. Personal samplers monitor the immediate environment of the subject permitting a more accurate assessment of personal exposure than occurs with area sampling. While airborne nicotine would be specific for ETS, problems exist in using it as a surrogate. Nicotine in ETS is principally in the gas phase [15], while nicotine in MS is almost exclusively in the particulate phase. Therefore, in ETS, nicotine would be serving as a surrogate for gas-phase components only.

Additionally, nicotine in ETS decays more rapidly than other gas-phase components [22], in large part due to its adsorption onto surfaces. It is likely that, once smoking has stopped in a room, the adsorbed nicotine will be slowly released back into the atmosphere. If this occurs, a low level of airborne nicotine may be present in an area where smoking had not occurred for some time giving an inaccurate representation of total ETS exposure.

The ratio of RSP/nicotine has been discussed as a possible monitor for ETS in ambient environments, and in particular as a means for quantifying the ETS-specific RSP [45]. Laboratory studies have given an average ratio of 13.4. Using values for RSP and nicotine from field surveys, it has been concluded that the relationship between these two materials is too variable to use for predictive purposes [46].

The mutagenic properties of RSP have been used to assess exposure to ETS [26]. The principal problem with this approach is the interpretation of the results. The significance of the presence of airborne

mutagens has not been established nor have quantitative measures of retention of mutagenic materials been obtained. Because of these uncertainties, the measurement of airborne mutagenicity has provided little information in assessing exposure to ETS.

To date no single material satisfies the criteria as a marker for ETS. Consequently, it has not been possible quantitatively to assess the external dose of ETS a non-smoker receives.

#### *Assessment of internal dose.*

As an assessment of exposure to ETS, biomarkers can serve as surrogates for the internal dose received. Criteria have been proposed that an effective marker should satisfy [47]:

1. It should be tobacco-specific in order to be certain of its origin.
2. It should have a long half-life so that it serves as an index of exposure over an extended period of time.
3. The marker should give a valid indication of the health risks of exposure.
4. Analytical techniques should be available that can reliably and conveniently measure the low levels of the marker present in non-smokers exposed to ETS.

Biomarkers of ETS exposure have been measured in biological fluids of humans. Several biomarkers have been utilised with varying degrees of success in the assessment of exposure to ETS, including nicotine and cotinine in saliva, blood, and urine, DNA and protein adducts in blood, and mutagenic activity in urine. From these results, investigators have drawn conclusions about exposure, risk of disease, and mortality.

When interpreting studies in which biomarkers have been used to assess exposure or risk, a number of factors must be considered [48]. Data on variation among individuals in absorption, metabolism (including bioactivation and detoxication), kinetics, distribution, excretion, binding to macromolecules and cellular repair must be evaluated. In the

2023380111

use of biomarkers for assessment of exposure to ETS and assessment of potential health risks, such considerations have not been employed consistently.

Biomarkers such as nicotine, or one of its metabolites, cotinine, in body fluids have been used to assess internal exposure to ETS [41,48–50]. In general, salivary and urinary cotinine provide the best relationship with self-reported exposure to ETS [47,51]. Levels of cotinine in body fluids tend to correlate directly with the number of smokers in the household, the number of hours of exposure, the number of smokers among acquaintances, and are higher in non-smokers married to smokers than in those married to non-smokers.

Nevertheless, significant limitations exist in the use of nicotine or cotinine to assess exposure to ETS. At best, levels of nicotine or cotinine are useful qualitatively to assess exposure. Too many limitations exist for them to be considered quantitative dosimeters from which risk can be estimated [52]. In virtually all studies reported, single samples are taken in the assessment of exposure. Such values are an index of exposure at a specific point in time and do not represent the cumulative exposure that would be required properly to evaluate exposure to ETS. Importantly, the vast majority of nicotine in ETS is in the gas phase while nicotine in MS is predominantly in the particulate phase [22,53]. Therefore, values for nicotine or cotinine in body fluids represent the inhalation of physically different materials in the two exposure groups making their comparative use questionable. Additionally, gas-phase nicotine and particulate-phase nicotine decay at different rates under experimental conditions [22]. Levels of nicotine or cotinine in body fluids provide no information on exposure to other chemicals, particularly those in the particulate phase which are believed to have the most relevance to potential adverse health effects.

It was once thought that one of the attractive features of using nicotine and cotinine as biomarkers was their tobacco-

specificity. Recent studies indicate that nicotine is not unique to tobacco. A number of vegetables in our diet have been shown to contain nicotine [54,55]. The fact that nicotine, and consequently cotinine, can arise from non-tobacco sources complicates the interpretation of the low-level values of these chemicals that are measured in the body fluids of non-smokers.

Cotinine is only one of a number of metabolites of nicotine and evidence is now indicating that nicotine-n-oxides or trans 3'-OH-cotinine, rather than cotinine, may be the most abundant metabolites of nicotine in the urine [56–58]. Cholerton et al., [56] report a larger coefficient of variation for cotinine than other nicotine-derived metabolites in the urine of smokers. Variations in the metabolic formation of cotinine among non-smokers would further confound the interpretation of cotinine levels.

Complicating this problem even further are pharmacokinetic factors. Nicotine appears to be metabolised at different rates in smokers and non-smokers [59–61]. The half-life of nicotine in plasma appears to be shorter for smokers than for non-smokers, therefore, the relative relationship of values between the two groups will differ depending upon the time of sampling.

Both intralaboratory and interlaboratory variations have been reported for urinary cotinine values [62,63], indicating that comparisons of values among laboratories should be made with caution. Such methodological considerations are of particular significance when values are low as is the case with exposure to ETS.

It seems evident that the measurement of cotinine in body fluids will likely provide misleading information regarding the quantitative exposure to ETS. Considering the factors discussed, a compelling argument can be made against using nicotine or cotinine values for either a quantitative comparison of exposure between smokers and those exposed to ETS or in an

attempt to assess the possible risk of exposure to ETS.

Urinary cotinine as a predictor of health risks of exposure to ETS should be used with caution [64]. Nevertheless, a risk assessment estimating ETS-related mortality has been made using such values. One study reported that urinary nicotine values in non-smokers were 0.7% of the level found in smokers [8] and the assumption made that there are premature deaths from the inhalation of ETS which may be approximately 0.7% of that due to active smoking resulting in 1,000 deaths a year in Great Britain and 4,000 deaths a year in the United States. No consideration was given to the limitations in the use of this marker. Additionally, the authors assumed that the relationship of dose-to-risk is linear between these two exposure extremes, an assumption that has not been shown to be valid. Clearly, this risk assessment is overly simplistic and confounded by a number of significant conceptual problems.

Wigle et al. [65], used values for urinary cotinine of active smokers and non-smokers exposed to ETS to assess the relative exposures of non-smokers to components of tobacco smoke that have been reported to be toxic. They concluded that persons exposed to ETS for 20 or more hours per week have exposures to six compounds that have been designated as known or probable human carcinogens which are at least 2% of those of active smokers and, for certain of these compounds, may be more than 20%. In arriving at these estimates, the authors made a number of assumptions which ignore the complexity of the exposure situation. In particular, SS was used as a surrogate for ETS. Such a premise is clearly inappropriate and invalidates any quantitative relationships that might be developed.

DNA and protein adducts have been utilised as biomarkers to assess internal exposure to ETS. Adducts are products derived from covalent reactions between chemicals and biological material such as

DNA and proteins. The formation of DNA adducts is reported to be associated with mutagenesis and carcinogenesis [66,67], and adducts are viewed as markers of the biologically effective dose of carcinogens in humans. Recent evaluation of the role of adducts in carcinogenesis indicates that the relationship may not be as direct as initially thought [68].

In spite of the lack of correlation between adduct levels and cancer in a number of studies [69-72] considerable interest continues in their use as molecular dosimeters for carcinogenesis. Although studies have started to examine their possible role in the assessment of exposure to ETS, little useful information currently exists in this context.

For many chemicals, adduct formation following metabolic activation is a necessary, but not sufficient, event to initiate carcinogenesis [73,74]. The formation of adducts may not occur on a region of the genome that is critical in the carcinogenic process. The role of DNA repair must also be considered [75,76]. The variability in repair capabilities in humans [77] will influence the level of adducts present in a tissue. Additionally, genetic polymorphism of drug metabolism in humans has been shown to result in wide interindividual capacities to activate carcinogens metabolically [78]. Because cancer is a multistage process, and because the level of adducts may be influenced by a multitude of factors including diet [79] it is thought to be unlikely that DNA adducts will provide precise quantitative dosimetry for predicting cancer risk [80], particularly where the level of adducts is as low as observed for ETS exposure. Another line of research in this area involves proteins as target molecules for adduct formation with the goal of serving as a surrogate for DNA adducts [81]. Because of its abundance, haemoglobin has been used to monitor adduct levels associated with exposure to tobacco smoke [82,83].

A potentially attractive aspect of the use of adducts as a dosimeter for ETS exposure, is that they may be useful in

monitoring exposure, at least qualitatively, on a more chronic basis than with other markers. To date, no tobacco-specific adduct has been identified that is capable of fulfilling this goal. Adducts of 4-amino-biphenyl-haemoglobin (4-ABP-Hb) and of benzo[a]pyrene diol epoxide-1-DNA (BPDE-1-DNA) in white blood cells have been compared in smokers and non-smokers [82-84]. While both 4-ABP and benzo[a]pyrene (BP) have been classified as carcinogenic, neither is tobacco-specific. Levels of 4-ABP-Hb adducts have, however, been used to distinguish smokers from non-smokers. The levels of 4-ABP-Hb adducts in non-smokers have been reported to be about one fifth the level found in smokers [82,83]. In one study, BPDE-1-DNA adducts were of little value in distinguishing the two groups [83]. Over a 48 h period, there was little consistency in the presence of adducts in smokers, with many smokers having no detectable levels. Additionally, there was no apparent correlation between the level of 4-ABP-Hb adducts and the level of BPDE-1-DNA adducts in either group.

Adducts of 4-ABP-Hb and 3-ABP-Hb have been measured in the blood of non-smokers with varying degrees of exposure to ETS as assessed by the presence or absence of detectable serum cotinine [82]. In non-smokers exposed to ETS, the 4-ABP-Hb levels were about 40-fold higher than the level of 3-ABP-Hb which in many subjects was below the limit of detection. Due to the lack of a clear cut effect of ETS exposure on 4-ABP-Hb adduct levels, and the inconsistent detectability of 3-ABP-Hb adducts, the usefulness of these markers to discriminate non-smokers exposed to ETS from those who are not exposed, appears questionable.

Recent studies indicate that the turnover of adducts may be more rapid than originally thought, limiting their usefulness to monitor chronic exposure. While the lifespan of haemoglobin is 120 days [81], levels of 4-ABP-Hb adducts in smokers returned to background levels in 6-8 weeks following cessation of smoking.

[82]. NNK is a tobacco-specific nitrosation product of nicotine that is present in MS and SS and has been classified as carcinogenic in animals [85]. Removal of adducts induced by the injection of NNK, has been examined in rats [69]. Rates of removal of different adducts in target tissues was variable and rapid, occurring within several days. These data indicate that NNK-induced adducts may not be useful as a dosimeter for tobacco smoke exposure.

A very sensitive method for examining the presence of adducts is  $^{32}\text{P}$  post-labelling. This technique provides a semi-quantitative estimate of the adduct level in a tissue. At present, there has been little application of this technique in assessing exposure to ETS. In spite of its sensitivity, there are limitations associated with the  $^{32}\text{P}$  post-labelling technique. It does not allow identification of the adduct, and basal levels of adducts are reported to increase with age, at least in animals [86]. Using this technique, no increase in DNA adducts was reported in monocytes of non-smokers heavily exposed to ETS [87].

In order to compare the potential risks of exposure to ETS with those reported for active smoking, an extrapolation from high-dose exposure to low-dose exposure is required. DNA adducts have been proposed as a means to do this. The relationship between external dose and biological dose, as assessed by DNA adducts, is dependent on the absorption, distribution, metabolism and excretion of the chemical of interest. The interpretation of biological dose using DNA adducts is influenced by several factors, including the location of the adducts on the genome as well as the mutagenic efficiency of the material, including the base that is modified and the effectiveness of the repair process. Additionally, for certain chemicals, the level of adduct formation is not linearly related to the dose administered. From the existing data, no absolute information is available relating the presence of adducts to a quantitative or qualitative assessment of exposure to ETS.

2023380113

As a measure of exposure to ETS, studies have been conducted on the capability of concentrated extracts from the urine of non-smokers and persons exposed to ETS to induce mutations in bacteria [88–94]. The rationale behind this approach is that the presence of mutagens in the urine may be an indication that the person has been exposed to chemicals that can ultimately induce cancer. Compared to the mutagenicity of the urine of smokers, activity in the urine of those exposed to ETS is quite low, variable and not always above background levels.

A number of problems exist with the studies attempting to relate urinary mutagenicity to ETS exposure. The experimental conditions of exposure to ETS have often been unrealistic in comparison to that occurring in the ambient environment. Methodological differences exist among studies possibly contributing to some of the inconsistencies. The studies have not always been controlled for the presence of dietary mutagens, an important confounding factor [95]. Importantly, the putative mutagens have not been identified. Finally, the biological significance of low-level mutagenicity in urinary concentrates has not been established. Due to these factors there is little reason, at present, to believe that urinary mutagenicity can be used to assess exposure to ETS or to assess risk to cancer [93].

#### Extrapolation models

An important consideration in the dose-response analysis of risk assessment is the extrapolation model used at the low-dose end of the curve. Traditionally, the linear non-threshold dose-response model has been used in the quantitative risk assessment of carcinogens. Current evidence brings this concept into question [96] and necessitates a rethinking of this process. The theory presented is that, at low doses, initiation may occur but unless exposure to high doses of promoters then occurs, tumours will not develop. This line of reasoning has considerable impact on the procedures used for analysing low-dose exposure as it relates to extrapolation of cancer risk from active smoking to exposure to ETS.

In spite of the distinct differences in dose received from active smoking and exposure to ETS, the extent of exposure to ETS and active smoking has been compared through the use of cigarette equivalents [21,40,43,46,47,65,97]. This approach attempts to convert exposure to ETS into an equivalent exposure from active smoking with the assumption that the risk from ETS exposure is proportionally comparable to the risk from active smoking. This procedure is an oversimplification of the exposure conditions and will provide potentially misleading information [3].

#### Pathophysiological consequences and implications

As indicated above, it is extremely difficult to extrapolate from active smoking to ETS exposure with any degree of reliability. Similarly, the data do not point to consistent evidence of pathophysiological consequences of ETS based on exposure and dose. Some examples will be presented to illustrate this point.

Several studies have reported that, functionally, smokers may have reduced ventilatory function at rest and a reduced exercise capacity with a greater oxygen debt accumulation [98–101]. For ETS-exposed non-smokers, the effects on ventilatory function and exercise capacity reductions are not consistent. While a few studies show some functional impairment, the majority do not. First of all, it is difficult to determine if the test situation mimics real-life exposure. The conditions to which subjects are exposed are often not relevant to ETS exposure. One study where subjects were passively exposed to cigarette smoke illustrates this point [102]. After drawing the puff through the apparatus consisting of a solenoid, capacity vessel and pump, the MS was discharged into the test room along with the SS. Therefore, the subjects were essentially breathing diluted quantities of the same constituents as an active smoker. The exposure conditions were also rather

202338014

extreme. Initial concentrations of particulate matter were  $>4\text{mg}/\text{m}^3$  and carbon monoxide levels were 24 ppm. After 2 h, the particulate concentration dropped to only  $2\text{mg}/\text{m}^3$ . Therefore, these conditions are not representative of ambient ETS exposure.

Even in this study [102], no change was found in the FEV<sub>1</sub> of the subjects at rest. When bicycle exercise was performed, the only change found was a slight increase in heart rate at two to five time points that was statistically significant but not biologically important.

Another problem in trying to identify possible effects of ETS on pulmonary function is the inaccurate or broad ranges of exposure as represented in either the ETS-exposed or -unexposed groups. Usual confirmation of ETS exposure or lack of active smoking is through questionnaires without chemical confirmation. No matter how limited chemical confirmation techniques are, questionnaires are less reliable. Most epidemiological studies involve spousal exposure and ignore whether smoking occurs in the home to any significant degree or whether spousal exposure is compounded by workplace or social exposure. Intuitively, it might be expected that smokers socialise with others who also smoke more often than do non-smokers. The other major consideration may be tied to the general health status or awareness of smoker households compared to non-smoker households. It would seem very important to match groups for diet and exercise as well as other health indicators.

#### *Functional studies*

In contrast to the studies reported on MS, it would appear that there is little agreement among studies as to the effects of ETS on pulmonary parameters. Even within studies, unexplainable peculiarities appear that raise questions of reliability. Certain age groups of particular populations are found to be affected where other population segments in the same study show increased pulmonary function capability. In a comprehensive review of

this subject the results of studies were regarded as being too variable to permit a conclusion concerning long-term ETS exposure and possible impaired respiratory health or pulmonary function in non-smoking adults [103].

Studies typically are further complicated by the possibility of suggestibility. Suggestibility is the reverse of the placebo effect. These studies are performed to determine the magnitude of the psychosomatic effect and hope to answer the question: "If the subject expects an adverse effect to occur, will this be reflected in a measurable response?" Here again, there is no good agreement. One study reports a 50% increase in airways resistance following a positive suggestion that the subject would be breathing a substance that may be irritating and make it harder to breath [104]. In another study, subjects who could easily tell whether or not they were breathing the smoke, were exercised at a level to increase minute ventilation to about 2.5 times resting ventilation. These subjects showed a dose-related response to sham or zero smoke, and two levels of ETS exposure [105]. The magnitude of change in pulmonary function parameters was minor in most cases and of no physiological significance. The experiment was flawed by the failure clearly to separate the psychological influence from the physiological effects and to establish any real controls, whereas the previously cited study [104] unquestionably separated the two components. Furthermore, in this study [105] it appeared that all smoke, including the MS generated by the smoking machines, was presented to the subjects.

The question of allergic response to tobacco smoke has been raised frequently, and was investigated by McDougall and Gleich [106], who reported that tobacco and tobacco smoke allergies were not demonstrable. It might thus be concluded that most of the apparent irritation in the presence of ETS is psychologically based.

When considering asthmatic patients, where active smoking has sometimes

2023380115

2023380116

been reported to be capable of triggering attacks, the evidence is not well established for ETS. Pulmonary function tests of asthmatics produced no change in expiratory flow rates. However methacholine challenge did produce a slight but significant increase in airway reactivity [107]. Other investigators studied the effects of ETS on asthmatics and found variable and inconclusive results in pulmonary function, but again found the increased reactivity to challenge; this time to histamine [108]. The results seem reasonable; however the regimen was not clearly stated. The mixing of MS and ETS may be a confounding problem of this study, as well. In summary, these results suggest a highly variable functional response to ETS even under laboratory conditions.

#### *Cancer types, locations and frequencies*

Use of tobacco products has been reported to be associated with cancers of various types and in various organ systems depending upon the tobacco product used. A review which addresses the comparisons between active smoking and exposure to ETS, concludes that more research needs to be done to demonstrate a strong association between ETS and cancer in the non-smoking population [109].

These authors begin with the hypothesis that the association between ETS and lung cancer must be possible based on the evidence from active smoking. They then examine the criteria set forth in the Surgeon General's report of 1964, and cite the inconsistencies in the results of both prospective and case-control studies. They make a specific point of the necessity for carefully documenting tumours using good histopathological techniques. In their own previously reported and unreported studies, they found that there is a preponderance of Kreyberg type I class tumours associated with smoking. In never-smokers, the preponderance of tumours are classified as Kreyberg type II. Within these categories the squamous cell type (type I) was predominant in smokers, "with lesser but

significant causative effect on the glandular type". In non-smokers, the predominant type is the glandular adenocarcinoma type II tumour. Other authors [110-111] suggest that ETS is limited to squamous cell types of tumours. If this is the case, the numbers of tumours potentially attributable to ETS would be very small considering the low incidence of this type of lung cancer in non-smokers. There is some support for squamous cell tumours being the most likely to be caused by ETS [112], quoted by the US Surgeon General [113]. In a closely monitored study in Olmsted County, Minnesota, Beard and his colleagues found that the incidence rate for squamous cell tumours dropped remarkably in the 1965-1974 period, presumably as smoking decreased. Small cell tumour incidence, also associated with smoking, decreased but not as dramatically. The incidence of adenocarcinoma continued to rise. There are several conclusions that can be drawn:

1. If Dalager et al. [110] and Pershagen et al. [111] are correct in concluding that squamous cell and small cell tumours are the predominant types associated with both smoking and exposure to ETS, then the risk of lung cancer from ETS is very small since this tumour is rare in non-smokers.
2. Since adenocarcinoma of the lung continued to rise in the Olmsted County study and is purported by some investigators to be the predominant type for ETS exposure, the association between ETS and adenocarcinoma is incorrect, meaning that some other cause is associated with the development of adenocarcinoma of the lung.
3. ETS may not, in fact, cause cancer of the lung at all, or if it does, perhaps it is associated with several types of tumours but not at a very high level.

Regardless of who is correct, more careful documentation is necessary of the histological types and incidence of lung tumours in order to determine an accurate and meaningful risk.

## Conclusions

Since ETS has not been adequately characterised, there are insufficient data on which to base a hazard analysis. Accordingly, there are not enough data available on which to base an exposure assessment for ETS. Due to the dynamic nature of ETS, it is impossible to relate ETS to MS chemically or physically. In the absence of this relationship, it is inappropriate to make any extrapolations from what is reported about the effects

of active smoking to possible effects of exposure to ETS. Therefore, any calculation of risk from exposure to ETS based on extrapolations from calculated risks of active smoking is, at best, not reliable and, most probably, of no value whatsoever. It is important, therefore, to consider ETS as a distinct entity, and further research is needed to test hypotheses based on valid protocols that meet the criteria established for the epidemiology of weak associations.

## References

1. Jenkins, R.A., Guerin, M.R.: General analytical considerations for the sampling of tobacco smoke in indoor air. In: Environmental Carcinogens: Methods of Analysis and Exposure Measurement. Vol. 9. Passive Smoking; O'Neill, I.K., Brunnemann, K.D., Dodet, S., Hoffmann, D. (eds). IARC Publications No. 81. International Agency for Research on Cancer, Lyon, France, 1987.
2. National Research Council, Environmental Tobacco Smoke-Measuring Exposures and Assessing Health Effects. National Academy Press, Washington, DC, 1986.
3. US Surgeon General; The Health Consequences of Involuntary Smoking; U.S. Department of Health and Human Services, Washington, DC, 1986.
4. Repace, J.L., Lowrey, A.H.: Risk assessment methodologies for passive smoking-induced lung cancer. *Risk Anal.* 1990; 10: 27-37.
5. Fong, P.: The hazard of cigarette smoke to nonsmokers. *J. Biol. Phys.* 1962; 10: 65-73.
6. Repace, J.L., Lowrey, A.H.: A quantitative estimate of nonsmokers' lung cancer risk from passive smoking. *Environ. Internat.* 1985; 11: 3-22.
7. Robins, J.: Risk assessment-exposure to environmental tobacco smoke and lung cancer. National Research Council, Environmental Tobacco Smoke-Measuring Exposures and Assessing Health Effects. National Academy Press, Washington, DC, 1986; pp. 294-337.
8. Russell, M.A.H., Jarvis, M.J., West, R.J.: Use of urinary nicotine concentrations to estimate exposure and mortality from passive smoking in non-smokers. *Brit. J. Addict.* 1986; 81, 275-281.
9. Dube, M.F., Green, C.R.: Methods of collection of smoke for analytical purposes. *Recent Adv. Tobacco Sci.* 1982; 8: 42-102.
10. Norman, V.: An overview of vapor phase, semivolatile and nonvolatile components of cigarette smoke. *Recent Adv. Tobacco Sci.* 1977; 3: 28-58.
11. National Research Council, Risk Assessment in the Federal Government: Managing the Process. National Academy Press, Washington, DC, 1983.
12. Baker, R.R., Proctor, C.J.: The origins and properties of environmental tobacco smoke. *Environ. Internat.* 1990; 16: 231-245.
13. Adams, J.D., O'Mara-Adams, K.J., Hoffmann, D.: Toxic and carcinogenic agents in undiluted mainstream and sidestream smoke of different types of cigarettes. *Carcinogenesis* 1987; 8: 729-731.
14. Benner, C.L., Bayona, J.M., Caka, F.M., et al.: Chemical composition of environmental tobacco smoke. 2. particulate-phase compounds. *Environ. Sci. Technol.* 1989; 23: 686-698.
15. Eatough, D.J., Hansen, L.D., Lewis, E.A.: The chemical characterization of environmental tobacco smoke. In: Environmental Tobacco Smoke. Proceedings of the International Symposium at McGill University 1989; Ecobichon, D.J., Wu, J.M. (eds). Lexington Books, DC Heath and Co., Lexington, Mass., 1990; 3-50.
16. Guerin, M.R., Higgins, C.E., Jenkins, R.A.: Measuring environmental emissions from tobacco combustion: sidestream cigarette smoke literature review. *Atmosph. Environ.* 1987; 21: 291-297.
17. Sakuma, H., Kusama, M., Munakata, S. et al.: The distribution of cigarette smoke components between mainstream and sidestream smoke. II Acidic components. *Brit.*

2023380112

- Tabakforsch. Internat.* 1984a; 12: 63-71.
18. Sakuma, H., Kusama, M., Yamaguchi, K., et al.: The distribution of cigarette smoke components between mainstream and sidestream smoke. II. Basic components. *Berl. Tabakforsch. Internat.* 1984b; 12: 199-209.
19. Sakuma, H., Kusama, M., Yamaguchi, K., Sugawara, S.: The distribution of cigarette smoke components between mainstream and sidestream smoke. III. Middle and higher boiling components. *Berl. Tabakforsch. Internat.* 1984c; 12: 251-258.
20. Hoffmann, D., Adams, J.D., Haley, N.J.: Reported cigarette smoke values: a closer look. *Amer. J. Public Health* 1983; 73: 1050-1053.
21. Brunnemann, K.D., Adams, J.D., Ho, D.P.S., Hoffmann, D.: The influences of tobacco smoke on indoor atmospheres: 2. Volatile and tobacco-specific nitrosamines in main- and sidestream smoke and their contribution to indoor pollution. *Proceedings of the 4th Joint Conference on Sensing of Environmental Pollutants*, New Orleans, 1977, American Chemical Society, 1978, pp. 876-880.
22. Tang, H., Richards, G., Gunther, K. et al.: Determination of gas phase nicotine and 3-ethenylpyridine, and particulate phase nicotine in environmental tobacco smoke with a collection bed-capillary gas chromatography system. *J. High Resolution Chrom. Chrom Commun.* 1988; 11: 775-782.
23. Ingbrethsen, B.J., Sears, S.B.: Particle size distribution measurements of sidestream cigarette smoke. *39th Tobacco Chemists' Research Conference*, Montreal, Canada, 1985.
24. Pritchard, J.N., Black, A., McAughey, J.J.: The physical behavior of sidestream tobacco smoke under ambient conditions. *Environ. Tech. Lett.* 1988; 9: 545-552.
25. Vu-Duc, T., Huynh, C-K.: Sidestream tobacco smoke constituents in indoor air modeled in an experimental chamber-polycyclic aromatic hydrocarbons. *Environ. Internat.* 1989; 15: 57-64.
26. Carson, J.R., Erikson, C.A.: Results from survey of environmental tobacco smoke in offices in Ottawa, Ontario. *Environ. Tech. Lett.* 1988; 9: 501-508.
27. Lofroth, G., Ling, P.L., Agurell, E.: Public exposure to environmental tobacco smoke. *Mutation. Res.* 1989; 202: 103-110.
28. Oldaker, G.B. III, Conrad, F.C. Jr.: Estimation of effect of environmental tobacco smoke on air quality within passenger cabins of commercial aircraft. *Environ. Sci. Technol.* 1987; 21: 994-999.
29. Proctor, C.J., Warren, N.D., Bevan, M.A.J.: An investigation of the contribution of environmental tobacco smoke to the air in betting shops. *Environ. Tech. Letters* 1989a; 10: 333-338.
30. Proctor, C.J., Warren, N.D., Bevan, M.A.J.: Measurement of environmental tobacco smoke in an air-conditioned office building. *Present and Future of Indoor Air Quality*, Bieva, C.J., Courtois, Y., Govaerts, M. (eds). Elsevier Science Publishers, BV, 1989b: 169-172.
31. Stenlik, G., Richter, O., Altmann: Concentration of dimethylnitrosamine in the air of smoke-filled rooms. *Ecotoxicol. Environ. Safety* 1982; 6: 495-500.
32. Sterling, T.D., Mueller, B.: Concentrations of nicotine, RSP, CO and CO<sub>2</sub> in nonsmoking areas of offices ventilated by air recirculated from smoking designated areas. *Am. Ind. Hyg Assoc. J.* 1988; 49: 423-426.
33. Cummings, K.M., Markello, S.J., Mahoney, M.C., Marshall, J.R.: Measurement of lifetime exposure to passive smoke. *Amer. J. Epidemiol.* 1989; 130: 122-132.
34. Friedman, G.D., Petitti, B.D., Bawol, D.D.: Prevalence and correlates of passive smoking. *Amer. J. Public Health* 1983; 73: 401-406.
35. McCarthy, J., Spengler, J., Chang, B-H. et al.: A personal monitoring study to assess exposure to environmental tobacco smoke. *Proc. 4th Int. Conf. Indoor Air Quality & Climate*, West Berlin, 1987; 2: 142-146.
36. Wu-Williams, A.H., Samet, J.M.: Environmental tobacco smoke: Exposure-response relationships in epidemiological studies. *Risk Anal.* 1990; 10: 39-48.
37. Coglin, J., Hammond, S.K., Gann, P.H.: Development of epidemiological tools for measuring environmental tobacco smoke exposure. *Amer. J. Epidemiol.* 1989; 130: 696-704.
38. Repace, J.L.: Indoor concentrations of environmental tobacco smoke: models dealing with effects of ventilation and room size. *IARC Publication* 1987; 81: 25-41.
39. Arundel, A., Sterling, T., Weintraub, J.: Never smoker lung cancer risks from exposure to particulate tobacco smoke. *Environ. Internat.* 1987; 13: 409-426.
40. Hinds, W.C., First, M.W.: Concentrations of nicotine and tobacco smoke in public places.

2023380118

2023380119

- New Engl. J. Med. 1975; 292: 844-845.
41. Hoffmann, D., Haley, N.J., Adams, J.D., Brunnemann, K.D.; Tobacco sidestream smoke: Uptake by nonsmokers. Prev. Med. 1984; 13: 608-617.
  42. Miesner, E.A., Rudnick, S.N.I., Hu, F.C., et al.; Particulate and nicotine sampling in public facilities and offices. JAPCA 1989; 39: 1577-1582.
  43. Muramatsu, M., Umemura, S., Okada, T., Tomita, H.; Estimation of personal exposure to tobacco smoke with a newly developed nicotine personal monitor. Environ. Res. 1984; 35: 218-227.
  44. Muramatsu, M., Umemura, S., Fukui, J., et al.; Estimation of personal exposure to ambient nicotine in daily environment. Int. Arch. Occup. Environ. Health 1987; 59: 545-550.
  45. Leaderer, B.P.; Assessing exposures to environmental tobacco smoke. Risk Anal. 1990; 10: 19-26.
  46. Oldaker, G.B. III., Crouse, W.E., Depinto, R.M.; On the use of environmental tobacco smoke component ratios. Present and Future of Indoor Air Quality. Bieva, C.J., Courtois, Y., Govaerts, M. (eds). Elsevier Science Publishers, BV, 1989: 287-290.
  47. Jarvis, M.J.; Application of biochemical intake markers to passive smoking measurement and risk estimation. Mutation Res. 1989; 222: 101-110.
  48. Coultas, D.B., Howard, C.A., Peake, G.T., et al.; Salivary cotinine levels and involuntary tobacco smoke exposure in children and adults in New Mexico. Ann. Rev. Respir. Dis. 1987; 136: 305-309.
  49. Griffith, J., Duncan, R.C., Hulka, B.S.; Biochemical and biological markers: Implications for epidemiological studies. Arch. Environ. Health 1989; 44: 375-381.
  50. Greenberg, R.A., Haley, N.J., Etzel, R.A., Loda, F.A.; Measuring the exposure of infants to tobacco smoke. Nicotine and cotinine in urine and saliva. New Engl. J. Med. 1984; 310: 1075-1078.
  51. Jarvis, M.J., Russell, M.A.H., Feyerabend, C., et al.; Passive exposure to tobacco smoke: saliva cotinine concentrations in a representative population sample of non-smoking school children. Brit. Med. J. 1985; 291: 927-929.
  52. Idle, J.R.; Titrating exposure to tobacco smoke using cotinine: a minefield of misunderstandings. J. Clin. Epidemiol. 1990; 43: 313-317.
  53. Eudy, L.W., Thorne, F.A., Heaven, D.L., et al.; Studies on the vapor-particulate phase distribution of environmental nicotine. Presented at the 39th Tobacco Chemists Research Conference, Montreal, Canada, October 2-5, 1985.
  54. Castro, A., Monji, N.; Dietary nicotine and its significance in studies on tobacco smoking. Biochem. Arch. 1986; 2: 91-97.
  55. Sheen, S.J.; Detection of nicotine in foods and plant materials. J. Food. Sci. 1988; 53: 1572-1573.
  56. Cholerton, S., Ayesh, Idle, J.R., Smith, R.L.; The pre-eminence of nicotine N-oxidation and its diminution after carbimazole administration. Brit. J. Clin. Pharmacol. 1988; 26: 652P-653P.
  57. Neurath, G.B., Pein, F.G.; Gas chromatographic determination of trans-3'-hydroxycotinine, major metabolite of nicotine in smokers. J. Chromatograph 1988; 431: 216-221.
  58. Parviaainen, M.K., Barlow, R.D.; Assessment of exposure to environmental tobacco smoke using a high-performance liquid chromatographic method for the simultaneous determination of nicotine and two of its metabolites in urine. J. Chromatog. 1988; 431: 216-221.
  59. Haley, N.J., Sepkovic, D.W., Hoffmann, D.; Elimination of cotinine from body fluids: Disposition in smokers and nonsmokers. Amer. J. Public Health 1989a; 79: 1046-1048.
  60. Kyerematen, G.A., Damiano, M.D., Dvorchik, B.H., Vessell, E.S.; Smoking-induced changes in nicotine disposition: Application of a new HPLC assay for nicotine and its metabolites. Clin. Pharmacol. Ther. 1982; 32: 769-780.
  61. Sepkovic, D.W., Haley, N.J., Hoffmann, D.; Elimination from the body of tobacco products by smokers and passive smokers. JAMA 1986; 256: p. 863.
  62. Biber, A., Scherer, G., Hoepfner, I., et al.; Determination of nicotine and cotinine in human serum and urine: An interlaboratory study. Toxicol. Lett. 1987; 35: 45-52.
  63. Letzell, H., Fischer-Brandies, A., Johnson, L.C., et al.; Measuring problems in estimating the exposure to passive smoking using the excretion of cotinine. Toxicol. Lett. 1987; 35: 35-44.
  64. Haley, N.J., Colosimo, S.G., Axelrad, C.M., et al.; Biochemical validation of self-reported

- exposure to environmental tobacco smoke. *Environ. Res.* 1989b; 49: 127-135.
65. Wigle, D.T., Collishaw, N.E., Kirkbride, J.; Exposure of involuntary smokers to toxic components of tobacco smoke. *Can. J. Public Health* 1987; 78: 151-154.
  66. Peikonen, O., Vahakangas, K., Nebert, D.W.; Binding of polycyclic aromatic hydrocarbons to DNA: Comparison with mutagenesis and carcinogenesis. *J. Toxicol. Environ. Health* 1980; 6: 1009-1020.
  67. Wogan, G.N., Gorelick, N.J.; Chemical and biochemical dosimetry of exposure to genotoxic chemicals. *Environ. Health Perspect.* 1985; 62: 5-18.
  68. Henderson, R.F., Bechtold, W.E., Bond, J.A., Sun, J.D.; The use of biological markers in toxicology. *CRC Crit. Rev. Toxicol.* 1989; 20: 65-82.
  69. Belinsky, S.A., White, C.A., Devereux, T.R., Anderson, M.W.; DNA adducts as a dosimeter for risk estimation. *Environ. Health Perspec.* 1987; 76: 3-8.
  70. Phillips, D.H., Grover, P.L., Sims, P.; The covalent binding of polycyclic hydrocarbons to DNA in the skin of mice of different strains. *Internat. J. Cancer* 1978; 22: 487-494.
  71. Randerath, E., Mittal, D., Randerath, K.; Tissue distribution of covalent DNA damage in mice treated dermally with cigarette 'tar': preference for lung and heart DNA. *Carcinogenesis* 1988; 9: 75-80.
  72. Randerath, E., Miller, R.H., Mittal, D. et al.; Covalent DNA damage in tissues of cigarette smokers as determined by  $^{32}\text{P}$ -postlabeling assay. *J. Natl. Cancer Inst.* 1989; 81: 341-347.
  73. Bradley, M.O., Sina, J.F., Erickson, L.C.; Measurements of chemical interactions with DNA. In: *Mechanisms and Toxicity of Chemical Carcinogens and Mutagens*, W.G. Flamm, R.J. Lorentzen (eds). Vol. XII of *Advances in Modern Environmental Toxicology*, M.A. Mehilman (ed). Princeton Scientific Publishing Co., Inc., Princeton, NJ, 1985: pp. 99-127.
  74. Pegg, A.E.; Alkylation and subsequent repair of DNA after exposure to dimethylnitrosamine and related carcinogens. *Reviews in Biochemical Toxicology*, Vol. 5, Hodgson, E., Bend, J.R., Philipot, R.M. (eds). Elsevier Science Publishing Company, New York 1983: 83-133.
  75. Doerjer, G., Bedell, M.A., Oesch, F.; DNA adducts and their biological relevance. In: *Mutations in Man*, G. Obe (ed). Springer Verlag, Heidelberg 1984: 20-34.
  76. Pegg, A.E.; Formation and subsequent repair of alkylation lesions in tissues of rodents treated with nitrosamines. *Arch. Toxicol.* 1980; Suppl. 3: 55-68.
  77. Oesch, F., Aulmann, W., Platt, K.L., Doerjer, G.; Individual differences in DNA repair capacities in man. *Arch. Toxicol.* 1987; suppl. 19: 172-179.
  78. Harris, C.C., Autrup, H., Vahakangas, K., Trump, B.F.; Interindividual variation in carcinogen activation and DNA repair. *Genetic Variability in Responses to Chemical Exposure*. Banbury Report 1984; 16: 145-154.
  79. Jahnke, G.D., Thompson, C.L., Walker, M.P. et al.; Multiple DNA adducts in lymphocytes of smokers and nonsmokers determined by  $^{32}\text{P}$ -postlabeling analysis. *Carcinogenesis* 1990; 11: 205-211.
  80. Harris, C.C., Weston, A., Willey, J.C. et al.; Biochemical and molecular epidemiology of human cancer: indicators of carcinogen exposure, DNA damage, and genetic predisposition. *Environ. Health Perspec.* 1987; 75: 109-119.
  81. Skipper, P.L., Tannenbaum, S.R.; Protein adducts in the molecular dosimetry of chemical carcinogens. *Carcinogenesis* 1990; 11: 507-518.
  82. MacIure, M., Katz, R.B.-A., Bryant, M.S. et al.; Elevated blood levels of carcinogens in passive smokers. *Amer. J. Public Health* 1989; 79: 1381-1384.
  83. Perera, F.P., Santella, R.M., Brenner, D. et al.; DNA adducts, protein adducts and sister chromatid exchange in cigarette smokers and nonsmokers. *J. Nat. Cancer Inst.* 1987; 79: 449-456.
  84. Bryant, M.S., Skipper, P.L., Tannenbaum, S.R., MacIure, M.; Hemoglobin adducts of 4-aminobiphenyl in smokers and nonsmokers. *Cancer Res.* 1987; 47: 602-608.
  85. Hecht, A.A., Chen, C.B., Ohmori, T., Hoffmann, D.; Comparative carcinogenicity in F344 rats of the tobacco-specific nitrosamines, N'-nitrosomornicotine and 4-(methylnitrosamino)-1-(3-pyridyl)-1-butanone. *Cancer Res.* 1980; 40: 298-302.
  86. Randerath, K., Reddy, M.V., Fisher, R.M.; Age- and tissue-related DNA modifications in untreated rats: detection by  $^{32}\text{P}$ -postlabeling assay and possible significance for tumor induction and aging. *Carcinogenesis* 1986; 7: 1615-1617.
  87. Holz, O., Krause, T., Scherer, G. et al.;  $^{32}\text{P}$ -postlabelling analysis of DNA adducts in monocytes of smokers and passive smokers. *Int. Arch. Occup. Environ. Health* 1990; 62: 299-303.

2023380121

88. Bos, R.P., Theuws, J.L.G., Henderson, P.T.; Excretion of mutagens in human urine after passive smoking. *Cancer Lett.* 19; 85-90.
89. Husgafvel-Pursiainen, K., Sorsa, M., Engstrom, K., Einisto, P.; Passive smoking at work: biochemical and biological measures of exposure to environmental tobacco smoke. *Int. Arch. Occup. Environ. Health* 1987; 59; 337-345.
90. Mohtashamipour, E., Muller, G., Norpoth, K. et al.; Urinary excretion of mutagens in passive smokers. *Toxicol. Lett.* 1987; 35; 141-146.
91. Putzrath, R.M., Langley, D., Eisenstadt, E.; Analysis of mutagenic activity in cigarette smokers' urine by high performance liquid chromatography. *Mutation Res.* 1981; 85; 97-108.
92. Scherer, G., Westphal, K., Biber, A. et al.; Urinary mutagenicity after controlled exposure to environmental tobacco smoke (ETS). *Toxicol. Lett.* 1987; 35; 135-140.
93. Scherer, G., Westphal, K., Adlikofer, F., Sorsa, M.; Biomonitoring of exposure to potentially genotoxic substances from environmental tobacco smoke. *Environ. Internat.* 1989; 15; 49-56.
94. Sorsa, M., Einisto, P., Husgafvel-Pursiainen, K. et al.; Passive and active exposure to cigarette smoke in a smoking experiment. *J. Toxicol. Environ. Health* 1985; 16; 523-534.
95. Sasson, I.M., Coleman, D.T., LaVoie, E.J. et al.; Mutagens in human urine: effects of cigarette smoke and diet. *Mutation Res.* 1985; 158; 149-157.
96. Albert, R.; Carcinogen risk assessment. *Environ. Health Perspect.* 1989; 81; 103-105.
97. Russell, M.A.H., West, R.J., Jarvis, M.J.; Intravenous nicotine simulation of passive smoking to estimate dosage to exposed non-smokers. *Brit. J. Addict.* 1985; 80; 201-206.
98. Dockery, D.W., Speizer, F.E., Ferris, B.G. Jr. et al.; Cumulative and reversible effects of lifetime smoking on simple tests of lung function in humans. *Am. Rev. Respir. Dis.* 1988; 137; 286-292.
99. US Surgeon General; Smoking and Health, Report of the Advisory Committee to the Surgeon General of the Public Health Service, US Department of Health, Education and Welfare, Public Health Service, 1964.
100. US Surgeon General; Consequences of Smoking, US Department of Health, Education and Welfare, Washington, DC, 1973.
101. US Surgeon General; The Health Consequences of Smoking: A Reference Edition-Selected Chapters from 1971 through 1975 Reports. US Department of Health, Education and Welfare, Washington, DC, 1976.
102. Pimm, P.E., Shepard, R.J., Silverman, F.; Physiological effects of acute passive exposure to cigarette smoke. *Arch. Environ. Health* 1978; 33; 201-213.
103. Witorsch, P.; Effects of ETS exposure on pulmonary function and respiratory health in adults. Environmental Tobacco Smoke: Proceedings of the International Symposium at McGill University 1989, Ecobichon, D.J., Wu, J.M. (eds), Lexington Books, DC Heath and Co., Lexington, Mass., 1990; 169-186.
104. Urcn, R.B., Silverman, F., Corey, P. et al.; Does suggestibility modify acute reactions to passive cigarette smoke exposure? *Environ. Res.* 1988; 47; 34-47.
105. Kotses, H., Rawson, J.C., Wigal, J.K., Creer, T.L.; Respiratory airway changes in response to suggestion in normal individuals. *Psychosomatic Med.* 1987; 49; 536-541.
106. McDougall, J.C., Gleich, G.J.; Tobacco allergy-Fact or fancy? *J. Allergy Clin. Immunol.* 1976; 57; 237.
107. Wiedemann, H.P., Mahler, D.A., Loke, J. et al.; Acute effects of passive smoking on lung function and airway reactivity in asthmatic subjects. *Chest* 1986; 98; 180-185.
108. Knight, A., Breslin, A.B.X.; Passive cigarette smoking and patients with asthma. *Med. J. Australia* 1985; 142; 194-195.
109. Wynder, E.L., Kabat, G.C.; Environmental tobacco smoke and lung cancer: A critical assessment. In: Indoor Air Quality. H. Kasuga (ed), Springer-Verlag, Berlin, 1990; pp. 5-15.
110. Dalager, N.A., Williams-Pickle, L., Mason, T.J. et al.; The relation of passive smoking to cancer. *Cancer Res.* 1986; 46; 4807-4811.
111. Pershagen, G., Hrubeczk, Z., Swensson, C.; Passive smoking and lung cancer in Swedish women. *Amer. J. Epidemiol.* 1987; 125; 17-24.
112. Beard, C.M., Annegers, J.F., Woolner, L.B., Kurland, L.T.; Bronchogenic carcinoma in Olmsted County, 1935-1979. *Cancer* 1985; 55: 2026-2030.
113. US Surgeon General; Reducing the Health Consequences of Smoking. 25 Years of Progress: A Report of the Surgeon General, US Department of Health and Human Services, Washington, DC, 1989.

**2023380122**

**6**

2023380123

## APPENDIX II

### AIRCRAFT AIR QUALITY

- Calls for smoking restrictions and bans aboard commercial aircraft are based upon rhetoric regarding health and exposure claims, not substantive scientific data. Available research indicates that smoking bans will do little, if anything, to remedy common complaints of dry, stuffy air aboard commercial aircraft.
- In a comprehensive review of the relevant data on aircraft air quality and nonsmoker health, an environmental specialist concluded that "the available scientific evidence does not support the prohibition of smoking on commercial aircraft". He also noted that the available data "suggest that factors or substances" other than tobacco smoke "may be major contributors to subjective complaints of discomfort by passengers and flight crew."<sup>1</sup>
- Data from in-flight measurements of tobacco smoke constituents indicate that the contribution of tobacco smoke to cabin air quality is negligible, posing no demonstrable adverse health consequences for passengers or crew.<sup>2-7</sup> One of these studies, conducted in Europe in 1989, involved the most comprehensive testing and analysis of aircraft cabin air quality to date.<sup>2</sup> The results indicate that total exposure to tobacco smoke aboard aircraft is "rather small and insignificant in

2023380124

comparison to total life exposure to air pollution." The researchers concluded that any possible health effects were "not likely to have been elicited" by such exposures aboard flights. They also noted that irritation and annoyance commonly attributed to such exposures may have been "potentiated by the low humidity, high temperature and high carbon dioxide levels found."

- Other studies reached similar conclusions. For example, the principal author of a 1987 study of a commercial airline in the U.S. noted that the typical amount of tobacco smoke in no-smoking sections of the aircraft is so small that it would take 224 hours, or more than 9 days of non-stop flying, to reach the exposure equivalent of the nicotine in a single cigarette.<sup>3</sup>
- A study of flight attendants during transoceanic flights measured body fluid levels of nicotine and concluded that the concentrations were so small that they were "unlikely to have physiologic effect."<sup>6</sup> Another study of flight attendants measured exhaled carbon monoxide (a tobacco smoke constituent) and reported that the concentration of smoke to which the attendants were exposed was "too low to alter significantly their expired air carbon monoxide levels."<sup>7</sup>

2023380125

- In 1989, an Australian specialist in occupational health and preventive medicine concluded that the available data "do not lend support to the hypothesis" that exposure to tobacco smoke "may present a risk to the health of cabin staff or passengers." He further observed that providing smoking and nonsmoking sections "meets the reasonable requirements of passengers."<sup>8</sup>
- Tobacco smoke, because it is easily seen, is readily blamed for passenger and crew discomfort. But other aspects of the cabin environment, including poor ventilation and the presence of carbon dioxide, ozone and low relative humidity, may create discomfort. For example, the President of the U.S. Aviation Safety and Health Association suggested that "the real culprit" is the lack of fresh air ventilation.<sup>9</sup> Listing a number of complaints ranging from headaches, dry throats and coughs to fatigue and dizziness, he concluded that "these symptoms are not related or caused by smoking aboard aircraft. Nor will a smoking ban of any length correct this fresh-air deficiency."
- Restrictions and bans on smoking aboard commercial aircraft have been imposed despite the lack of convincing data suggesting that tobacco smoke may affect cabin air quality or the health of nonsmokers during flights. A case in point is the 1989 decision by the United States Congress to ban smoking

2023380126

aboard flights of six hours or less, meaning the virtual prohibition of all smoking since few domestic flights last that long. A brief history of this prohibition suggests the largely political motivation behind the ban.

- In 1987, the U.S. Department of Transportation (DOT) rejected a proposed ban recommended by the National Academy of Sciences<sup>10</sup> because it was not supported by data associating health effects and tobacco smoke exposures aboard aircraft.<sup>11</sup> Nonetheless, the next year, Congress voted to impose a temporary ban on smoking aboard domestic flights of two hours or less. The DOT subsequently issued a request for proposals to monitor in-flight exposures to tobacco smoke and other indoor air constituents<sup>12</sup> for the purpose of aiding Congressional deliberations on this subject. However, Congress decided not to wait for the results of the study, which became available several months after it had voted for the ban.
- The final DOT report<sup>13</sup> contained data for selected constituents from tobacco smoke and other sources collected aboard commercial flights, as well as a theoretical health risk assessment based upon the data. The data on tobacco smoke constituents suggest that individuals seated in nonsmoking sections are exposed to extremely low levels of those

2023380127

constituents. These data would seem to justify the assumption that separate seating minimizes the nonsmoker's exposure to cigarette smoke. However, the data also indicate extremely high levels of carbon dioxide from passenger respiration on the majority of all flights, which in turn suggests the possibility of poor ventilation and poor air quality regardless of the presence or absence of cigarette smokers.

- In a recent presentation on the DOT study, one of the principal scientists responsible for the study recommended that "the first question I would ask is whether or not Congress knew that the ETS results were not strongly compelling prior to the study's release and as a result preempted the use of the results in the deliberations on whether or not to make the ban permanent."<sup>14</sup>

2023380128

#### REFERENCES

1. Holcomb, L., "Impact of Environmental Tobacco Smoke on Airline Cabin Air Quality," Environ. Technol. Letters 9: 509-514, 1988.
2. Malmfors, T., et al., "Air Quality in Passenger Cabins of DC-9 and MD-80 Aircraft," Environ. Technol. Letters 10: 613-618, 1989.
3. Oldaker, G. and F. Conrad, "Estimation of Effect of Environmental Tobacco Smoke on Air Quality Within Passenger Cabins of Commercial Aircraft," Environ. Sci. Technol. 21: 994-999, 1987.
4. Muramatsu, M., et al., "Estimation of Personal Exposure to Tobacco Smoke with a Newly Developed Nicotine Personal Monitor," Environ. Res. 35: 218-227, 1984.
5. Muramatsu, M., et al., "Estimation of Personal Exposure to Ambient Nicotine in Daily Environment," Arch. Occup. Environ. Health 59: 545-550, 1987.
6. Foliart, D., et al., "Passive Absorption of Nicotine in Airline Flight Attendants," New Eng. J. of Med. 308(18): 1105, 1983.
7. Duncan, D. and P. Greaney, "Passive Smoking and Uptake of Carbon Monoxide in Flight Attendants," J. of the American Med. Association 251(20): 120-21, 1984.
8. Crawford, W., "Environmental Tobacco Smoke in Airliners--Health Issues," Aerospace: 12-17, July, 1989.
9. Trask, P., "Smoke Gets in Your Aisles," Wall Street Journal, Monday, September 21, 1987.
10. Committee on Airliner Cabin Air Quality, Board on Environmental Studies and Toxicology, Commission on Life Sciences, National Research Council, The Airline Cabin Environment: Air Quality and Safety, Washington, D.C., National Academy Press, 1986.
11. U.S. Department of Transportation, Airliner Cabin Air Quality: Report to Congress, U.S. DOT, February, 1987.
12. U.S. Department of Transportation, Solicitation, Offer and Award, August 5, 1988.
13. Nagda, N., et al., Airliner Cabin Environment: Contaminant Measurements, Health Risks, and Mitigation Options, Report

2023380129

No. DOT-P-15-89-5, Prepared by GEOMET Technologies, Inc., Germantown, Maryland, for the U.S. Department of Transportation, Washington, D.C., December, 1989.

14. Baker, S., "Health Risks to Airliner Occupants: Exposures to Environmental Tobacco Smoke and Other Pollutants" Presentation, ILSI Risk Science Institute, Brookings Institution, June 14, 1990.

2023380130

**2023380131**

### Author Index

Adlkofer, F.	6, 11, 13, 15, 16, 17, 21, 23, 24, 26
Arundel, A.	16, 17
Aviado, D.	25, 26
Badre, R.	6, 10
Baker, R.	2, 4
Baker, S.	33, 34
Bayer, C.	6, 11
Bergman, H.	7, 12
Biber, A.	13, 14
Bombick, D.	19, 20
Bos, R.	18, 19, 20
Carson, J.	5, 9
Castro, A.	13, 15
Claxton, L.	18, 20
Coultas, D.	7, 12
Cox, B.	5, 9
Crawford, W.	31, 34
Cummings, K.	13, 14
Curvall, M.	13, 14
Duncan, D.	5, 9, 29, 30, 34
Foliart, D.	6, 10, 29, 30, 34
Girman, J.	5, 9
Godish, T.	6, 11
Good, B.	6, 11
Haley, N.	13, 14, 24, 26
Hattemer-Frey, H.	6, 12
Hendel-Kramer, A.	6, 11
Hinds, W.	6, 10
Hoepfner, H.	21, 23
Holcomb, L.	29, 34
Holz, O.	21, 23
Hosein, R.	5, 9
Hugod, C.	6, 11
Husgafvel-Pursiainen, K.	18, 19, 20, 22, 23
Idle, J.	13, 14
Itani, S.	13, 14
Jenkins, R.	6, 10
Johnson, L.	13, 14
Kasuga, H.	21, 23
Kirk, P.	5, 9
Lee, P.	13, 14
Lehnert, G.	6, 10
Letzel, H.	13, 14
Löfroth, G.	2, 4, 18, 20
Malmfors, T.	29, 34

2023380132

Martin, F.	21,23
Matsushita, H.	18,20
McAughey, J.	7,12
Moghissi, A.	7,12
Mohtashamipur, E.	18,20,19
Muramatsu, M.	6,10,29,34
Nagda, N.	32,34
Nitta, H.	5,9
Nystrom, C.	2,4
Oldaker, G.	5,6,9,10,29,30,34
Perera, F.	21,23
Piade, J.	2,4
Pritchard, J.	7,11
Proctor, C.	2,4,5,6,9,11
Quackenboss, J.	5,10
Rawbone, R.	2,4
Scherer, G.	2,4,19,20,21,23
Schievelbein, H.	6,10
Schwartz, S.	13,15
Sega, K.	6,11
Shimizu, Y.	6,11
Spengler, J.	5,10
Stebbins, J.	7,12
Stehlik, G.	6,11
Sterling, T.	5,9
Sonnenfeld, G.	18,20
Sorsa, M.	22,23
Thompson, C.	6,10
Trask, P.	31,34
Upfal, M.	7,12
Yocom, J.	5,9
Wald, N.	13,14
Wallace, L.	6,12
Watts, R.	13,14

2023380133

**A**

**2023380134**

## THE ORIGINS AND PROPERTIES OF ENVIRONMENTAL TOBACCO SMOKE

Richard R. Baker and Christopher J. Proctor  
BAT(UK&E) Ltd., Research & Development Centre, Southampton, SO9 1PE, U.K.

EJ 8906-186M (Received 26 June 1989; accepted 2 December 1989)

Environmental tobacco smoke (ETS) is formed from cigarettes as sidestream and exhaled mainstream smoke diffuse into ambient air. Detailed studies are reviewed which describe how sidestream smoke is formed, its acceleration away from the cigarette and its chemical properties. As the smoke streams diffuse into the atmosphere they become greatly diluted and physical and chemical changes occur. A quarter of the material in sidestream particles evaporates, so that ETS nicotine is virtually entirely in the vapour phase, and the particles shrink. As cigarettes are smoked, the levels of ETS components rise and then fall exponentially due to air exchange and deposition of smoke particles onto surfaces. The decay of ETS also depends on the particular component, with nicotine decaying faster than other substances. In real-world environments, ETS is found along with chemicals and particles from many sources. Studies are reviewed which quantify the contribution of ETS to various indoor air environments. These include determination of the ETS proportion of total respirable particles, measurement of nicotine as a specific ETS marker, and comparisons of chemicals present in matched smoking and nonsmoking environments. The ETS contribution of volatile organic compounds in air is much less than that from other sources. The review emphasises the need for tobacco specific analytes to be used as ETS markers and/or to apportion ETS particulate matter from total particulate matter in the atmosphere.

### INTRODUCTION

Environmental Tobacco Smoke, ETS, has received considerable attention in recent years. ETS is the complex mixture of chemicals found in air as a specific result of tobacco smoking (Nystrom and Green 1986). Some reports have claimed that exposure to ETS can be harmful to the health of nonsmokers (USSG 1986; NRC 1986; ISCSH 1988). This issue has been discussed by scientists and epidemiologists for over a decade and although knowledge has increased over this period, it is still the subject of scientific controversy (Manel 1987; Uberla 1987).

In order to assess ETS properly it is necessary to understand some of its chemical and physical properties, and to ascertain the concentrations of ETS present in typical environments (Repace 1987b; Proctor et al. 1989a).

The aims of this paper are to describe the origins of ETS through an understanding of the combustion processes occurring within a cigarette, how it builds up and then decays in indoor air, and some of its properties. This paper considers ETS in relation to cigarettes, rather than that originating from cigars and pipes.

### THE ORIGINS OF ETS

ETS results from a combination of sidestream smoke (that which is released from the lit end of the cigarette), and exhaled mainstream smoke (that exhaled by the smoker after drawing on the cigarette), both greatly diluted by the ambient air.

#### Fresh sidestream smoke

Sidestream smoke is defined as all the smoke generated by a cigarette that is not mainstream. Side-

2023380135

stream smoke is made up of the sidestream plume which is emitted from the burning zone during both the puff and smoulder periods in an upwards direction (because of buoyancy), the smoulder stream which escapes from the mouth end of the cigarette during smoulder, and gases which diffuse out of the tobacco rod by diffusion during both the puff and smoulder periods (Lipp 1965; Hoegg 1972; Baker 1982). It has been estimated that the sidestream plume contributes about 95% to the total sidestream smoke (Hoegg 1972).

Physical and chemical aspects of the sidestream plume in the vicinity of the burning zone of a smouldering cigarette are illustrated in Fig. 1. (In both Figs. 1 and 2, % v/v is % volume/volume. It should be noted that 1 % v/v = 10 mL/L). The data in this figure were measured in a variety of experiments previously described (Baker 1982; Robinson 1987). The position of the sidestream smoke plume was obtained by carefully photographing the smouldering cigarette under controlled air flow conditions around the cigarette. The position of the gas phase plume was obtained in a series of experiments in which small sampling probes were placed around the cigarette and connected directly to a mass spectrometer. The temperature distribution was measured using small thermocouples placed around the burning zone. The velocity distribution was measured using a laser Doppler velocimeter technique.

The gas phase temperature distribution outside the cigarette is very similar to that obtained earlier by Neurath et al. (1966). The positions of the smoke and gas phase plumes have also been confirmed using a Schlieren optical method described by McRae and Jenkins (1987).

In the smoulder period between the puffs, a natural convection flow of air around the burning zone in an upwards direction (because of buoyancy) sustains burning but at a much lower intensity than during the puff. Little change occurs to the external temperature and oxygen distributions when the puff is taken (Baker 1982), indicating that the natural convection stream around the burning zone and into the sidestream plume is only slightly affected by the influx of air during the puff. The combustion processes occurring on the surface of the burning zone in the convection stream proceed independently of those inside.

The main products in the gas plume are carbon monoxide, carbon dioxide, hydrogen and water — the concentration distribution of carbon monoxide outside the burning zone during smoulder is shown in Fig. 1 and the forms of the profiles of the other gases and oxygen depletion are similar. The carbon

monoxide plume originates some 3 to 4 mm in front of the paper burn line, as do the external temperature contours. The carbon monoxide concentration immediately above the burning zone is higher than that just inside, and a similar situation exists for carbon dioxide. Thus, sidestream carbon monoxide and carbon dioxide are not formed only by the combustion products diffusing out of the burning zone — some must be formed on the external surface from reactions with the oxygen convected around the coal.

Fig. 2 illustrates the variations during the smoking cycle of the gas concentrations of oxygen, carbon dioxide, carbon monoxide and hydrogen at a specific point in the sidestream gas plume. This point is situated 1 mm above the surface of the burning zone, and 3 mm in front of the paper burn line. The plume hydrogen and carbon monoxide concentrations at this point increase during the puff while the carbon dioxide concentration falls. At the end of the puff, the level of all three products increases for about one second. This variation is very similar to that found inside the centre of the burning zone (Baker 1981), and is due to the outward diffusion of those products formed inside the burning zone. The small rise in carbon monoxide and fall in carbon dioxide during the puff are due, at least partly, to the carbonaceous reduction of carbon dioxide to the monoxide, which occurs as the temperature in the interior of the burning zone increases as the puff progresses. When the puff ends, the product formation-transmission balance inside the burning zone is interrupted, resulting in a local build-up of gases in their formation regions. These diffuse into the sidestream to deplete the local build-up.

In contrast to the gas phase plume, the sidestream smoke plume originates 0.4 mm behind the paper burn line, becoming visible at temperatures below about 150°C (Fig. 1). This is the approximate position of the tobacco pyrolysis and distillation region inside the cigarette (Baker 1981). Inside the cigarette in this region, a concentrated organic vapour is formed. During the smoulder period much of it will diffuse radially out of the cigarette through the partially degraded cigarette paper, although some will also diffuse axially towards the mouth end of the cigarette to form the smoulder stream. As the vapour diffuses through the paper to the outside, it is subjected to a sudden temperature decrease and dilution. These conditions favour the formation of relatively small aerosol particles compared to mainstream particles.

Since the sidestream plume is the major contributor to ETS, the rate at which sidestream smoke is transported away from the cigarette into the atmos-

2023380136

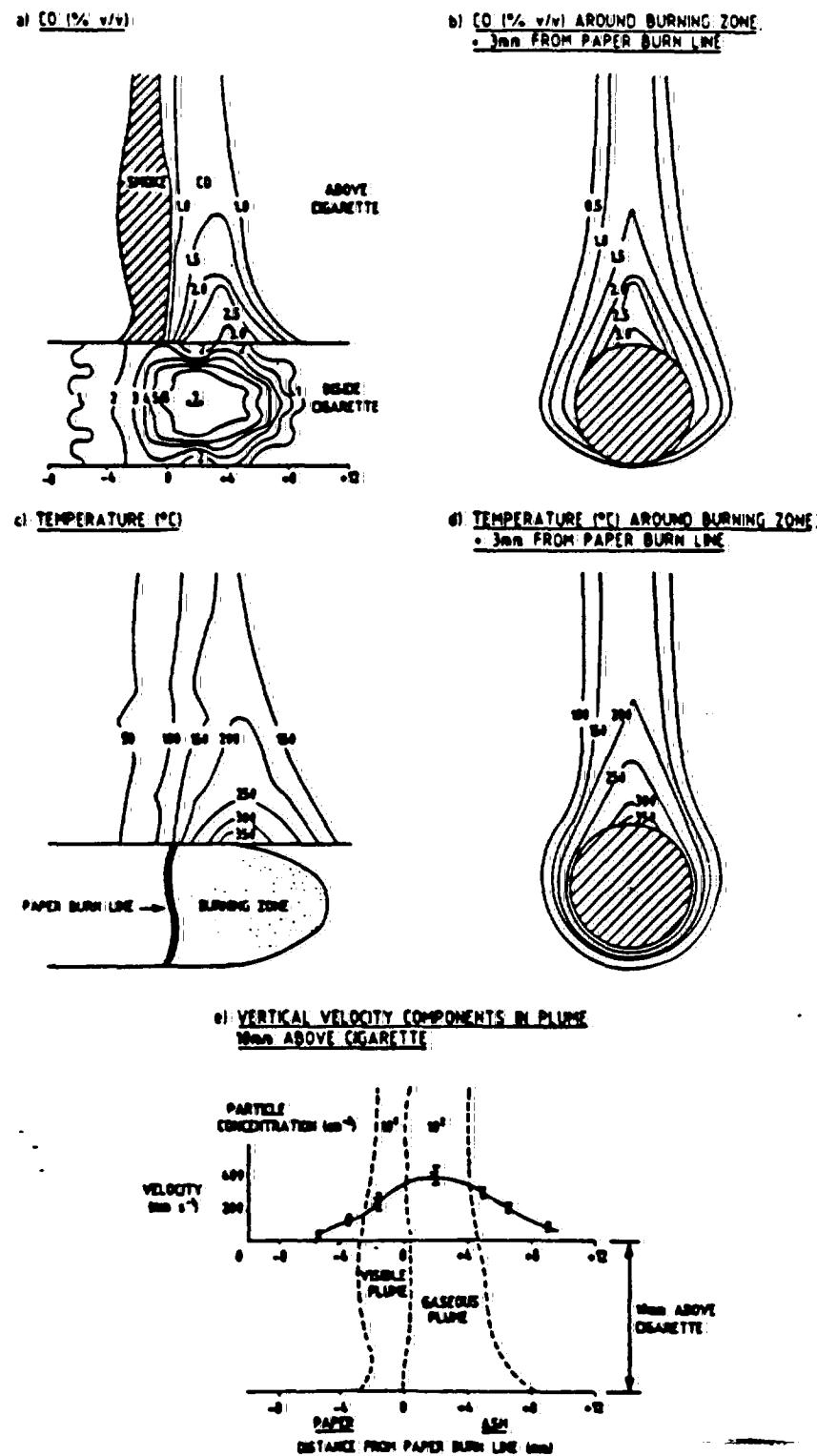


Fig. 1. Concentrations, temperatures and velocities in sidestream plume during smoulder (Baker 1982; Robinson 1987).

2023380137

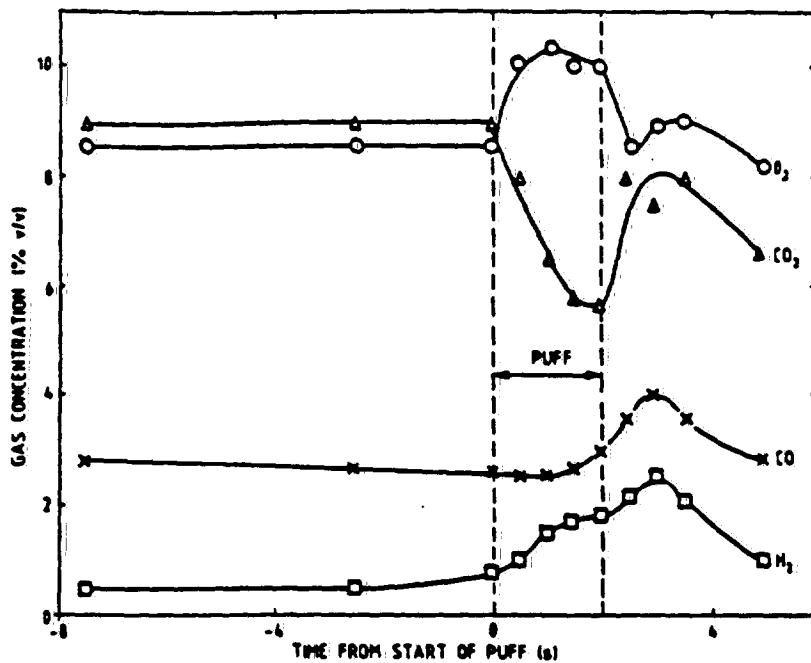


Fig. 2. Variation with time of sidestream gas concentration 1 mm vertically above cigarette and +3 mm from paper burn line.

sphere ultimately determines the rate of build-up of ETS components. A detailed determination of the velocity distribution of the sidestream plume has been made using a laser Doppler velocimeter described by Robinson (1987). In this technique, two laser beams are focused on a given point in the plume and produce an interference pattern at their point of intersection. Aerosol particles carried in the plume scatter light from the interference pattern and characteristics of this light can be used to calculate the velocity of the particles in the plume. The results indicate that at a given distance above the cigarette there is a distribution of velocity. That obtained at 10 mm above the cigarette is shown in Fig. 1(e) together with an estimate of the particle concentrations in the gas and smoke plumes. The peak velocity of  $410 \text{ mm s}^{-1}$  occurs in the gaseous plume, 2 mm in front of the paper burn line. The velocities in the smoke plume are generally less than half of this peak velocity.

Measurements at different distances above the cigarette show that the plume is accelerating as it rises above the cigarette (Fig. 3). This acceleration is accompanied by a falling temperature of the plume. The falling temperature of the plume with distance above the cigarette is accompanied by increasing density. This, along with the observed acceleration, means that there is a substantial increase in mass flow rate

in the plume with distance above the plume. This is brought about by air being radially drawn into the buoyancy driven, natural convection sidestream plume as it moves upwards. Detailed calculations and mathematical modelling have confirmed that this occurs (Robinson 1987; Robinson 1988).

Also shown in Fig. 3 are two previous measurements of the velocity of the sidestream smoke plume, that of Neurath et al. (1966) and that of Ayer and Yeager (1982). Both their values were single point measurements, i.e., they did not show any variation of velocity with distance above the cigarette. Clearly the actual flow structure of the sidestream plume is more complex than these two earlier measurements imply.

In general the same chemicals present in mainstream smoke are also present in sidestream smoke, though their relative yield per cigarette is highly dependent on the compound considered. Some typical sidestream/mainstream ratios are shown in Table 1 (Baker 1981; Guerin 1987; Kluus and Kuhn 1982; Sakuma et al. 1983, 1984a, 1984b; Norman et al. 1983; Umemura et al. 1986). These ratios were obtained from different studies using different tobaccos, cigarette types, methods of collecting the sidestream smoke, and air movement conditions around the cigarette. These differences will affect the results, reflected by the quoted range for a given compound in

2023380138

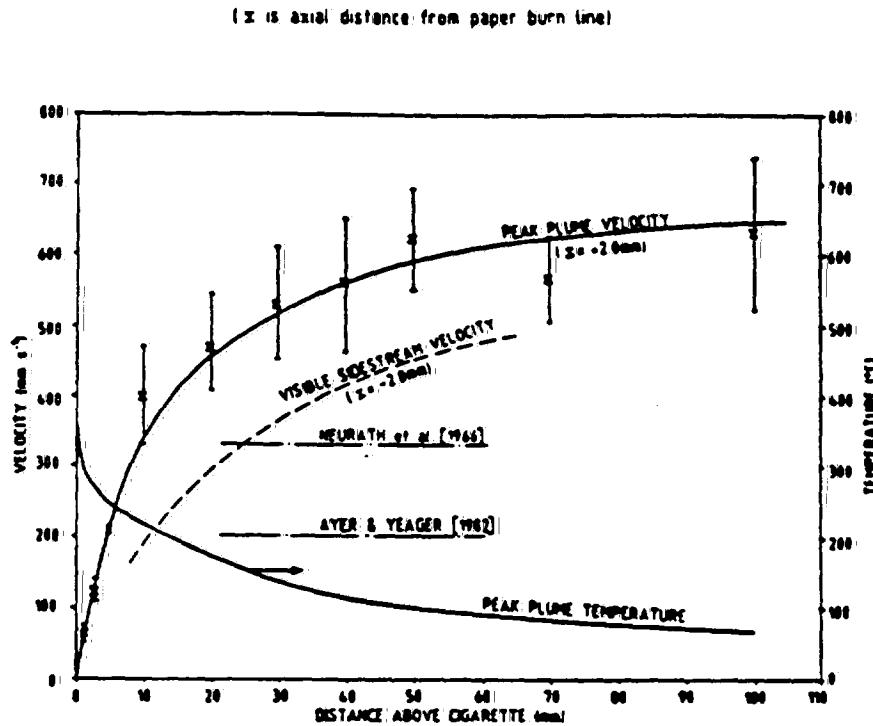


Fig. 3. Measured flow development in the sidestream plume (Robinson 1987).

Table 1. Some typical sidestream/mainstream (SS/MS) yield ratios.

Smoke Component	SS/MS
Hydrogen cyanide	0.06 - 0.5
Succinic acid	0.4 - 0.6
Hydroquinone	0.7 - 1.0
Neophytadiene	1.1 - 1.8
Phenol	1.6 - 3.0
Nicotine	1.9 - 3.3
Acetic acid	1.9 - 3.9
Carbon monoxide	2.5 - 4.7
Benzo(a)pyrene	2.7 - 3.4
Nitric oxide	3 - 13
Limonene	4 - 12
Toluene	5.6
Carbon dioxide	8 - 11
Acrolein	8 - 13
Pyrrole	9 - 14
Naphthalene	17
Pyridine	10 - 20
Water	30
Ammonia	44 - 170
Nitrogen	>270

the table. However, the data do show that there is a very wide range of ratios, varying from 0.06 - 0.5 for hydrogen cyanide to over 270 for molecular nitrogen formed chemically from the tobacco. Of course, one to four times more tobacco is burnt in smoulder than during puffing, depending on the cigarette parameters. Thus, some of the ratios in Table 1 (e.g., phenol and nicotine), simply reflect the proportions of tobacco burnt to the two smoke streams. However, there are many substances which distribute themselves so predominantly to one stream or the other that the reason cannot be due to differences in tobacco consumption. The reasons lie in the different conditions of temperature and mass transfer rates existing in the cigarette burning zone during smoulder and puffing, and the exact mechanism by which different components are formed or released from the tobacco.

During a puff, air is drawn into the peripheral regions of the burning zone, solid phase temperatures in excess of 900°C are reached and it is largely the periphery of the cigarette that burns (Baker 1981). When the puff ceases, the periphery of the burning

2023380136

zone cools rapidly to about 600°C; air is converted into the back of the burning zone and the axial portion of the tobacco rod is preferentially consumed during about the first twenty seconds of smoulder. The temperatures in the centre of the burning zone are in the region of 800°C and only increase by 50 - 80°C as the puff progresses. The major combustion products, carbon dioxide, carbon monoxide and water, are formed in the high temperature (> 500°C) region of the burning zone. However, the vast majority of smoke species are formed by pyrolysis/distillation processes in a relatively low temperature (< 500°C) oxygen deficient region, just behind the combustion zone in the region of the paper burn line. The concentrated organic vapour so formed is drawn down the tobacco rod during the puff to the mainstream and largely diffuses radially out of the rod during smoulder to form the sidestream smoke.

Hydrogen cyanide is formed via decomposition of nitrates and amino acids. The predominance in the mainstream must reflect a high temperature formation mechanism with sufficient temperatures hardly being attained during smoulder. On the other hand, ammonia, which is formed from the reduction of nitrates and pyrolysis of glycine, is delivered predominantly to the sidestream. Sufficient temperatures must exist during the smoulder period for the pyrolytic generation of the ammonia. Vapour phase water is also delivered almost exclusively to the sidestream and is believed to be derived from oxygen reacting with pyrolytically-generated hydrogen as it diffuses into the sidestream plume (Johnson 1975). Thus, the exact ratio in Table 1 very much depends on the mechanistic origin of each component.

The pH of mainstream and sidestream smoke also differs, with sidestream smoke being generally more alkaline. For example, the pH of the mainstream smoke of a U.S. blended cigarette is typically in the range 6.0 to 6.2 and that of the sidestream is in the range 6.7 to 7.5 (Brunnemann and Hoffmann 1974). In this study, the pH was determined by passing smoke over a sensitive combination electrode connected to a pH meter. The observed sidestream and mainstream difference are due to the predominance of basic components in the sidestream, e.g., ammonia, pyridine and nicotine, and carboxylic acids and phenols in the mainstream smoke. (However, pH is a concept applicable to aqueous solutions and strictly speaking it is not meaningful to give too much significance to the pH of a suspension of aerosol particles).

The mean size of the aerosol particles in sidestream smoke is smaller than in mainstream smoke.

Thus, Okada et al. (1977), using a light scattering technique, reported mainstream particles to have a geometric number mean diameter of 0.18 µm and sidestream particles of 0.12 µm. The mean size of the mainstream particles is smaller than that reported in other studies using other measurement techniques, cited by Okada et al. (1977). However, the relative size distributions of the mainstream and sidestream particles are seemingly authentic. The different size distributions for the two smoke streams must reflect the different rates of cooling and levels of air dilution to which their precursor vapours are subjected.

In mainstream smoke, Browne and co-workers (1980), have shown that nicotine is almost entirely in the particulate phase. This also is true for fresh, concentrated sidestream smoke (Proctor 1988a), though nicotine seems to transfer rapidly to the vapour phase as the smoke stream ages and becomes diluted (Eudy et al. 1986; Eatough et al. 1986, 1989).

Sidestream smoke yields will be dependant upon the weight of tobacco burnt during smoking, the construction of the cigarette, and the way in which it is smoked. However, typically, a conventional cigarette will yield around 600 mg of CO<sub>2</sub>, 4.5 mg CO, 5 mg of nicotine, and 25 mg particulate matter (water and nicotine subtracted) per cigarette in sidestream smoke.

#### *Exhaled mainstream smoke*

Few authors have considered exhaled mainstream smoke (EMS) as anything other than a minor contributor to ETS (Nystrom and Green 1986). Work recently completed in our laboratory suggests that the role of exhaled mainstream smoke should be considered in more detail. A variety of smokers were studied under carefully controlled conditions, smoking three types of cigarettes. These were a typical filtered British flue-cured cigarette (15 mg mainstream particulate delivery), a typical filtered case flavoured U.S. blended cigarette (14 mg), and a low delivery (3 mg) filter ventilated flue-cured cigarette. For each experiment three subjects smoked one cigarette type in their normal manner in a 30 m<sup>3</sup> chamber. This was repeated on three occasions for each of the cigarette types. For each experiment the maximum concentrations were determined for particulate matter (as measured by a piezobalance, TSI 5000), nicotine (Tenax trapping, GC-MS analysis) and carbon monoxide (by non-dispersive infrared spectroscopy).

Subsequently, cigarettes were smoked by machine (set at average parameters to mimic the human smoking) with mainstream smoke exhausted from the room. Hence the ETS from sidestream smoke alone was

2023380140

Table 2. Mean contributions of exhaled mainstream smoke (EMS) to ETS.

Cigarette	Contribution of EMS (%) to:		
	ETS Carbon Monoxide	ETS Particulate Phase	ETS Nicotine
Flue-cured	11	43	7
US-blended	13	15	9
Filter Ventilated	3	20	1

compared to ETS produced by humans, including exhaled mainstream smoke.

Table 2 presents the summary data for the three cigarette types. It can be seen that exhaled mainstream smoke contributes little to the gas phase of ETS and that this contribution is dependant upon the mainstream delivery of the cigarettes studied. However, EMS does significantly contribute to the ETS particulate phase. It is thought that particulate matter is retained by the smoker to a greater extent than carbon monoxide, and hence it would be expected that the EMS particulate contribution would be less than observed (Creighton 1973). The results may then indicate that EMS particles are more stable (perhaps larger and containing more water) than fresh sidestream particles.

#### AGEING OF TOBACCO SMOKE

As sidestream and exhaled mainstream smoke diffuse into the atmosphere and away from the cigarette and smoker, they become ETS. The originally concentrated sidestream and exhaled mainstream smoke streams become greatly diluted, the sidestream smoke cools and accelerates (Figs. 1, 3), and various physical and chemical changes occur in the smoke.

A variety of studies (Eatough et al. 1986, 1987; Eudy et al. 1986; Hammond et al. 1987) have shown that the nicotine in ETS is almost entirely in the vapour phase. Since ETS nicotine originates almost entirely from sidestream smoke (see Table 2), then nicotine in the fresh sidestream particles must rapidly evaporate out of the particles as the smoke ages during initial dilution.

Studies by Pritchard and co-workers (Black et al. 1987; Pritchard et al. 1988) have also shown that matter is evaporated from fresh sidestream particles as they are diluted to form ETS. They loaded 1-iodohexadecane labelled with  $^{123}\text{I}$  onto cigarettes. This material has a boiling point of  $380^\circ\text{C}$ , typical of that of components found in smoke particles. When

fresh sidestream smoke was collected from the smouldering, loaded cigarette using a "fish-tail" chimney collection system described elsewhere (Proctor et al. 1988a) it was found that 5% of the sidestream radioactivity was found in the vapour phase and 95% in the particulate phase. On the other hand when the cigarette smouldered in a steel chamber of  $14 \text{ m}^3$  internal volume, 70% of the airborne radioactivity was found to be in the vapour phase, and subsequent radiochemical analysis indicated that there had been no chemical degradation of the 1-iodohexadecane in the environmental chamber. Thus, the material had evaporated out of the sidestream particles during dilution to form ETS.

Ingebretsen et al. (1985, 1986) have independently estimated that 20 to 30% of the original matter in sidestream particles is lost by evaporation during the ageing of ETS. This estimate was calculated from measurements of the number and sizes of sidestream particles diluted in a stirred  $0.5 \text{ m}^3$  stainless steel chamber. The size distribution measurements were made by Ingebretsen and Sears (1985) using a combination of an optical particle counter, an electrostatic mobility analyser, and a condensation nucleus counter. Sidestream smoke was introduced into the  $0.5 \text{ m}^3$  chamber and diluted, and the number and sizes of the particles monitored over six hours. The estimated initial mass loss by evaporation of the smoke particles during about the first hour of ageing resulted in ETS particles with a mean number diameter of  $0.098 \mu\text{m}$  at typical ETS concentrations (several  $\mu\text{g m}^{-3}$  particulate matter). This is equivalent to a mass median diameter of  $0.185 \mu\text{m}$ . As the ETS aged over the next few hours in the stirred chamber, there was a slow but gradual increase in mean mass median diameter: 20% increase over 12 hours. This is due to a combination of coagulation of particles and removal of smaller particles by deposition onto surfaces of the chamber. Surface deposition of ETS is the main route of removal in a static environment and

2023380141

is a function of particle size, mixing rate, room size, and shape.

In addition to physical changes, chemical changes also occur as ETS ages. Thus, for example, nitric oxide slowly oxidises over minutes and hours to nitrogen dioxide in ETS (Piadé and Fink 1987; Klus et al. 1987; Baker et al. 1988). However, these chemical changes, and indeed the particle size changes described above, will be far outweighed by the physical effects of air movement which will occur in real indoor environments. These effects are described in the next section.

#### BUILD-UP AND DECAY OF ETS CONSTITUENTS

As cigarettes are smoked in a room, the levels of ETS components in the room rise and then fall due to air circulation, room ventilation and, to a lesser extent, interactions of the ETS constituents such as deposition of smoke particles onto surfaces in the room. It is normally not practical to measure the dynamic build-up and decay of ETS constituents in real-life environments and for such information specially constructed environmental rooms are used. In such rooms, the temperature, relative humidity, air circulation rate, and fresh air input can be varied over a wide working range and the observed dynamic ETS levels related to room environmental conditions. A number of studies in such rooms has been published in recent years (Hoegg 1972; Cain and Leaderer 1981; Case 1985; Blake et al. 1986; Heavner et al. 1986; Ingebretsen et al. 1986; Black et al. 1987; Eatough et al. 1987; Hiller et al. 1987; Olander et al. 1987; Piadé and Fink 1987; Pritchard et al. 1988; Rawbone et al. 1987a, 1987b; Vu Duc and Huynh 1987; Baker et al. 1988) or in standard offices with controlled environments (Klus et al. 1987).

Typical results from one study (Baker et al. 1988) are illustrated in Fig. 4. In this study the ETS was produced from the sidestream smoke of nonventilated filter cigarettes containing flue-cured tobacco smoked under standard smoking machine conditions in a 30 m<sup>3</sup> chamber. The chamber walls and ceiling had an impervious painted plastic finish and the floor was constructed of heavy duty non-slip PVC. Further details of the chamber have been given elsewhere (Case 1985; Baker 1988). Room conditions and number of cigarettes smoked were varied over the following ranges: temperature, 15-30°C; relative humidity, 55-85%; room air circulation, 1-27 air changes per hour; fresh air, 8-25%; number of cigarettes, 2-8.

During an experiment, samples of air were continuously removed from the room and analysed for particulate matter (using a TSI Model 5000 piezobalance

fitted with a 3.5 µm filter situated in the room), carbon monoxide and carbon dioxide (using non-dispersive infrared spectroscopy), nitric oxide, and nitrogen dioxide (using chemiluminescence), hydrocarbons (using flame ionisation), and particle size and number distributions (LAS-X laser aerosol spectrometer). For nicotine determination, time weighted average values were obtained over the five minute periods by sampling the air through thermal desorption tubes containing Tenax and subsequent analysis by gas chromatography/mass spectrometry. Room temperature and relative humidity were also monitored during each experiment. The data from all the instruments were monitored on a microcomputer, taking data points every 30 seconds. All results presented are corrected for background readings.

Individual data points are included on some of the profiles in Fig. 4 to indicate the precision of measurement. The signal to noise ratio for most of the instruments was better than 20:1, although that for nitrogen dioxide and total hydrocarbons was 5:1 and 10:1 respectively, due to the analysers being operated at maximum sensitivity. The coefficients of variation of all the profiles over five replicate experiments were better than 5%.

In Figs. 4 and 5, gas concentrations are quoted in mg m<sup>-3</sup> or µg m<sup>-3</sup>. It is, however, common practice in studies on ETS to use the units 'parts by volume per million' (ppm), which is equivalent to µL/L in the metric system of units, especially for carbon monoxide levels — see, for example, the review by Repace (1987b). The exact conversion factor depends on gas density, and for carbon monoxide at room temperature and pressure, 1 mg m<sup>-3</sup> = 1.15 ppm.

All the measured ETS components in Fig. 4 reach a maximum concentration at the end of smoking and then decay exponentially at a first order rate, i.e., decay rate is proportional to component concentration, as indicated by linear log<sub>e</sub> concentration/time plots (not illustrated). The nicotine profile apparently reaches a plateau after it has decayed to about 20% of its maximum value. The profiles for particle number concentration and aerosol mass are almost exactly parallel, as are the profiles for carbon monoxide and carbon dioxide. The count median diameter of the ETS aerosol particles remained constant at 0.13 µm as the ETS built up and decayed over the 5000 seconds of the determination. This is larger than that reported by Ingebretsen and Sears (1985) in their 0.5 m<sup>3</sup> chamber, due to the laser aerosol spectrometer in the current study not measuring the small particles below 0.1 µm which were included in In-

2023380142

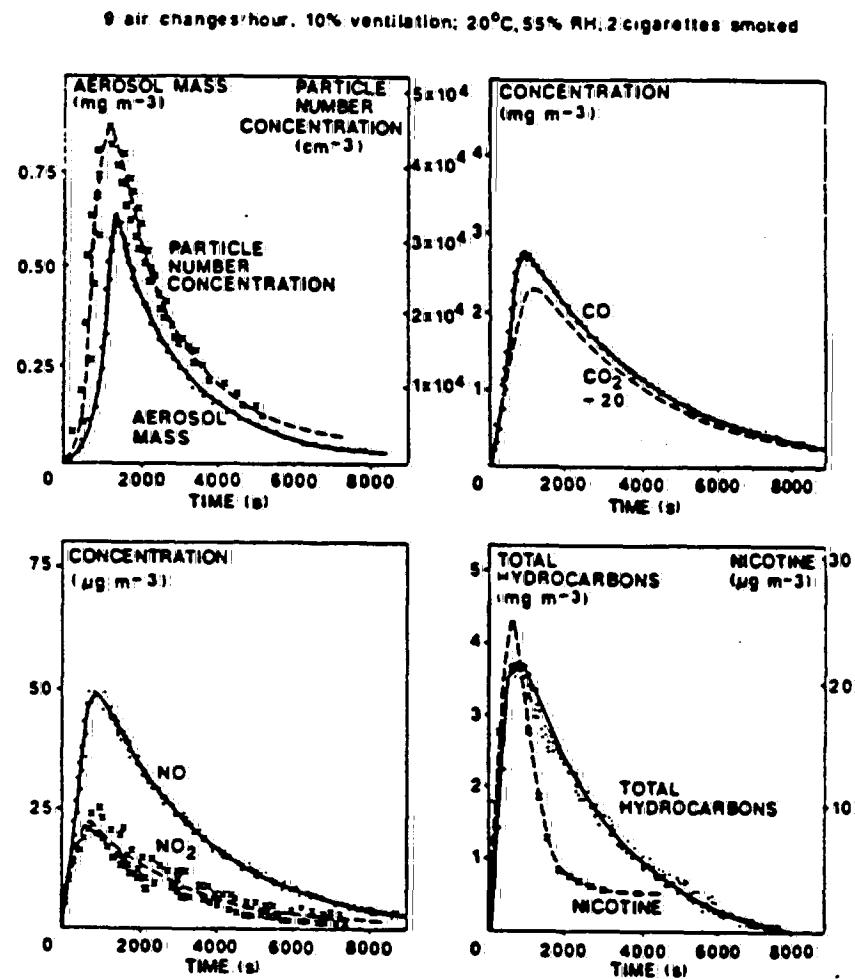


Fig. 4. ETS build-up and decay profiles.

gebretsen and Sears' combined technique described earlier.

For all the components studied, the peak ETS concentration (or time-weighted average for nicotine) increases linearly with the number of cigarettes smoked for 2 to 8 cigarettes. This linear relationship has been observed previously with up to 30 cigarettes smoked (Hoegg 1972; Case 1985; Blake et al. 1986). Using two types of experimental cigarettes with filter ventilation levels of 20 and 50%, Blake and co-workers (1986) have demonstrated the linear relationship for the following ETS components: carbon monoxide, nitric oxide, nitrogen dioxide, hydrogen cyanide, ammonia, formaldehyde, phenol, each of the three cresols, nicotine and aerosol particle mass.

The time taken for the peak ETS concentration to decay back to the background level depends on the environmental conditions. Fig. 5 illustrates the effect of air exchange rate (equal to room air circulation rate multiplied by the fraction of fresh air admitted to room) on the decay of carbon monoxide. Clearly, air movement has a large effect on the decay, as expected from mathematical considerations (Repace 1987a; Robinson 1988). The effect of air exchange rates on the half-life times of ETS components, obtained from profiles similar to those in Figs. 4 and 5 is illustrated in Fig. 6 over the sixteen sets of environmental conditions used in the study.

Half-life time is defined as the time to decay to half the value of the maximum concentration. Half-

2023380143

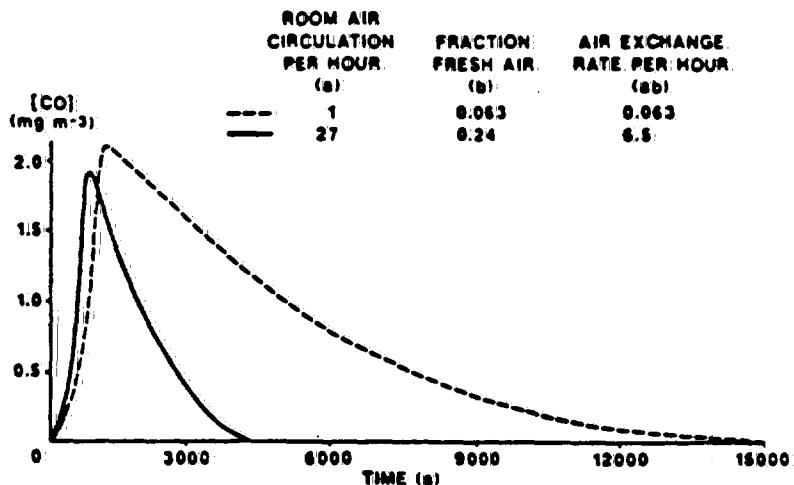
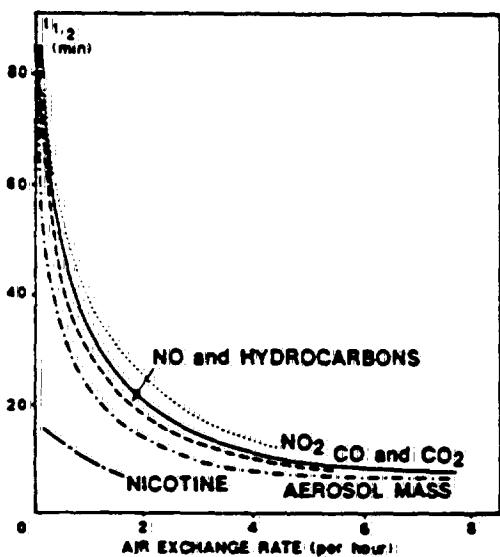


Fig. 5. ETS CO profiles for two room conditions.

Fig. 6. Variation of ETS  $t_{1/2}$  values with air exchange rate.

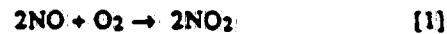
life times for nicotine were not obtained under all the conditions in the study, and in general are much smaller than the values for the other components, and much less influenced by the air movement. They are also less accurately defined, since they are based on average levels over five-minute periods rather than continuous measurements, and since nicotine is decaying relatively fast. Similar  $t_{1/2}$  values for nicotine can be obtained from the decay profiles reported in other studies: values of 10, 14 and 23 minutes from

Eudy and co-workers' profiles (1986) and 16 and 23 minutes from the profiles of Rawbone and co-workers (1987a). Variation of the environmental temperature (15–30°C) and relative humidity (55–85%) had a negligible effect on the  $t_{1/2}$  values (detailed results not illustrated).

For a given set of environmental conditions, the trends in Fig. 6 indicate that the relative  $t_{1/2}$  values of the ETS components are in the order:



The decay rates of the carbon oxides depend solely on the effects of air change rate and fresh air input (Piadé and Fink 1987) and their levels in ETS remain constant when there is no air movement (Heavner et al. 1986; Hiller et al. 1987). Where a component has smaller  $t_{1/2}$  values than carbon monoxide there must be some mechanism other than just air movement depleting its levels — chemical or physical. Nitric oxide  $t_{1/2}$  values are on average significantly less than those for carbon monoxide, and nitrogen dioxide  $t_{1/2}$  values are significantly higher, at the 90% confidence level. This is due to the conversion of nitric oxide to nitrogen dioxide in the ambient air, as postulated by others (e.g., Piadé and Fink 1987; Klus et al. 1987).



In fact, using the experimental data on ETS nitric oxide and nitrogen dioxide decay levels it can be shown that the rate constant for reaction [1] in ETS

2023380144

is almost an order of magnitude higher than that for the pure gas phase conversion but similar to that in the gas phase of mainstream smoke (Baker et al. 1988). Thus, in ETS, and the gas phase of mainstream smoke, the oxidation of nitric oxide is catalysed by substances which are not known.

The smaller values of  $t_{1/2}$  for aerosol mass than carbon monoxide must be due to deposition of the particles onto the surfaces in the room. The very much smaller values for nicotine could be due to adsorption of nicotine vapour onto the surfaces — since, as indicated in the preceding section, nicotine in ETS is largely in the vapour phase.

#### REAL-LIFE LEVELS OF ETS COMPONENTS

Although ETS originates from sidestream and exhaled mainstream smoke, the great dilution and other changes which these smoke streams undergo as they form ETS make their properties significantly different from those of ETS. Thus, the sidestream/mainstream ratios quoted in Table 1 can be misleading if used out of context. The important question is not the ratio of sidestream/mainstream but rather what is the concentration of the constituent in the indoor environment and how does it compare to levels from sources other than ETS. Studies based solely on observations of fresh sidestream, or highly and unrealistically concentrated ETS, should take into account the possible differences between these smokes and ETS found in real-life situations.

The previous sections have described how the concentration of ETS in a room will depend upon many factors such as the number of cigarettes smoked, how they were smoked and what type they were, and on the size and ventilation conditions in the room. The situation is further complicated in the real world by the fact that ETS is only one contributor to indoor air containing chemicals arising from multiple sources (Proctor et al. 1988b). All indoor air environments contain numerous chemicals as a result of emissions from, for example, building materials, furnishings, cooking and heating fuels, and consumer products (NRC 1981). Many of the chemicals associated with ETS will also be present as a result of such sources (Jenkins and Guerin 1984; Proctor et al. 1988a, 1989a, 1989b).

Hence, in order to determine potential exposures to ETS, it is essential to employ methods that allow a distinction between substances present as a result of tobacco smoking and substances present as a result of the other various sources. One approach is to identify a chemical marker that is specific, or at least indicative, to ETS (Haley et al. 1988).

Much early research used carbon monoxide concentrations to assess ETS levels (Sterling and Dimich 1982). However, there are many sources of carbon monoxide, such as gas cookers or heaters, open-fires, or motor vehicle emissions drawn in from outdoors, and it is not possible in real-life situations to segregate the ETS contribution from this background (NRC 1981; Girman and Traynor 1983; Haley et al. 1988).

Nicotine is far more specific, and had been used in recent years. The presence of nicotine in air is almost certainly indicative that tobacco smoking is, or has been taking place. Typically indoor levels range from 5 to 70  $\mu\text{g m}^{-3}$ .

However, it should be noted that the behaviour of nicotine in ETS is somewhat unusual when compared to many of the other constituents. Nicotine is primarily a vapour phase constituent in ETS, though a small portion (around 2%) will be found associated with the particulate phase (Eatough et al. 1989). Research has shown that the nicotine decays rapidly from an atmosphere in comparison even to other vapour phase constituents of ETS (Nystrom and Green 1986; Eatough et al. 1989 and Fig. 4 of the present paper). Moreover, it is also likely that some nicotine adsorbed onto walls and furnishings will be re-emitted. If this were to be the case, then areas where smoking had not taken place for some time might still exhibit a low level of airborne nicotine.

Even so, nicotine is currently the most useful specific chemical marker for ETS, and many field studies have utilised its measurement. For example, Thompson et al. (1989) found airborne nicotine concentrations in restaurants ranged from 0.5 to 37.2  $\mu\text{g m}^{-3}$  with a geometric mean of 3.5  $\mu\text{g m}^{-3}$ . In offices, Hammond et al. (1987) found personal exposures to nicotine (nonsmokers) ranging from 3.1 to 28.2  $\mu\text{g m}^{-3}$ , whilst Carson and Erikson, (1988) using fixed site monitoring, in a study of 31 offices in Ottawa found airborne nicotine geometric mean to be 7.2  $\mu\text{g m}^{-3}$  (range <1.2 to 69.7  $\mu\text{g m}^{-3}$ ). Research undertaken by our laboratory found similar levels of airborne nicotine with a median of 3.1  $\mu\text{g m}^{-3}$  (range 0.6 to 26  $\mu\text{g m}^{-3}$ ) in smokers' offices, 15.5  $\mu\text{g m}^{-3}$  (range 0.6 to 49.3  $\mu\text{g m}^{-3}$ ) in smoking-allowed train compartments, and 18  $\mu\text{g m}^{-3}$  (range 3 to 57  $\mu\text{g m}^{-3}$ ) in betting shops (Proctor et al. 1989a, 1989b).

Respirable suspended particulates (RSP) have also been determined in indoor air in relation to ETS. Virtually all ETS particulate matter will be in the respirable fraction (i.e., less than 3.5  $\mu\text{m}$  diameter) of airborne particulate matter.

2023380145

However, in real-world situations, ETS will seldom be the sole source of particulate matter. Hence it is important to try and estimate the proportion of particulate matter relating directly to ETS, rather than just measuring total particulate matter. One of the most frequently referenced papers is the work of Repace and Lowrey (1985). Their research suggested that a typical nonsmoker working in an office building in the U.S. would be exposed daily to average concentrations of particulate matter due specifically to ETS of  $242 \mu\text{g m}^{-3}$  (range 100 to  $1000 \mu\text{g m}^{-3}$ ). However, Samet et al. (1987) stated that surveys of indoor air quality based on measurement of total suspended particulate concentrations (such, presumably, as the Repace and Lowrey study) will not readily identify the excess mass indoors from environmental tobacco smoke.

Some researchers have attempted to develop more specific methodologies for the determination of the ETS proportion of total RSP. One method extracts respirable particulate matter collected on teflon coated filter pads, and subsequently analyses the extracts for their ultra-violet (UV) absorbance at 325 nm. By using a surrogate standard, 2,2<sup>1</sup>,4,4<sup>1</sup>-tetrahydroxybenzophenone, calibrated against ETS formed in controlled conditions, an estimate of the ETS contribution to particulate matter can be made (Thomas et al. 1989). This measure has been termed UV-RSP, and will often be an over-estimate, as smoke of the particulate matter in indoor air originating from sources other than ETS will also result in UV absorbance.

An alternative method, based on the same philosophy, analyses the methanol extracts of collected particulate matter for fluorescence (Thomas et al. 1989). Sample solutions are calibrated against dilute mainstream tobacco smoke solutions, and the method is suggested to have a greater sensitivity and selectivity than the UV-RSP measure.

A more specific particulate marker may be the compound solanesol. This substance is present in relatively high levels in tobacco and is transferred intact to sidestream smoke (Ogden and Maiolo 1988). However, it should be noted that different types of tobacco contain different levels of solanesol and hence the ambient concentration will be dependant not only on the number of cigarettes smoked but also, to some extent, on which brand of cigarettes was smoked.

Relatively few studies have published data using these particulate partitioning methods. Our data from offices, train compartments, and betting shops (Proctor et al. 1989a, 1989b) suggest that, although ETS does add to the particulate levels in indoor

environments, it may not always be the predominant source, and is unlikely to be the sole source. In smokers' offices a median RSP level of  $91 \mu\text{g m}^{-3}$  (range 33 to  $260 \mu\text{g m}^{-3}$ ) was found, though the corresponding UV-RSP data (the estimate of ETS contribution) gave a median value of  $24 \mu\text{g m}^{-3}$  (range 0.5 to  $75 \mu\text{g m}^{-3}$ ). This would suggest that in a relatively well ventilated office environment, ETS was contributing approximately, on average, 26% to the total RSP level. In smoking allowed-train compartments, a median RSP level of  $249 \mu\text{g m}^{-3}$  (range 71 to  $325 \mu\text{g m}^{-3}$ ) was identified. The corresponding UV-RSP data here were a median of  $70 \mu\text{g m}^{-3}$  (range 13 to  $110 \mu\text{g m}^{-3}$ ). This would suggest an approximate 28% contribution of ETS to total particulate matter. In betting shops (smoking allowed) the median RSP was  $284 \mu\text{g m}^{-3}$  (range 73 to  $767 \mu\text{g m}^{-3}$ ) whilst the median UV-RSP was  $109 \mu\text{g m}^{-3}$  (range 57 to  $610 \mu\text{g m}^{-3}$ ); an approximate ETS contribution of 38%. This research suggests the necessity for future studies to attempt to apportion ETS particulate matter from total particulate matter.

Although ETS contains many chemicals, research has been unable to identify good chemical markers apart from nicotine and ETS-specific RSP measures. Eatough et al. (1989) have suggested that 3-ethenylpyridine, myosamine, nitrous acid and pyridine may be possible markers for ETS gas phase, though more research is necessary to determine the validity of these substances.

An alternative approach to determining the contribution of ETS to an environment is to compare directly smoking and nonsmoking situations. In order to do this successfully, it is essential to match closely the factors impacting on the environments; for example smoking and nonsmoking situations should be of similar size, ventilation conditions, occupancy, furnishings, etc. This may be achieved by selecting sites within the same building, or by taking large numbers of sites for a particular environmental category. For example, Spengler et al. (1981) measured total RSP values in 80 homes to conclude that a smoker of 20 cigarettes per day would contribute  $20 \mu\text{g m}^{-3}$  to 24-hour indoor particulate concentrations. This type of approach is most usefully undertaken using ETS specific markers, but it may also identify differences in the levels of nonspecific chemicals associated with ETS. In the U.S. Environmental Protection Agency's Total Exposure Assessment Methodology (TEAM) study, levels of some airborne volatile organic chemicals (VOC) were suggested to be higher in smokers' homes than in nonsmokers' homes (Wallace et al. 1987). Other small studies have been unable to dis-

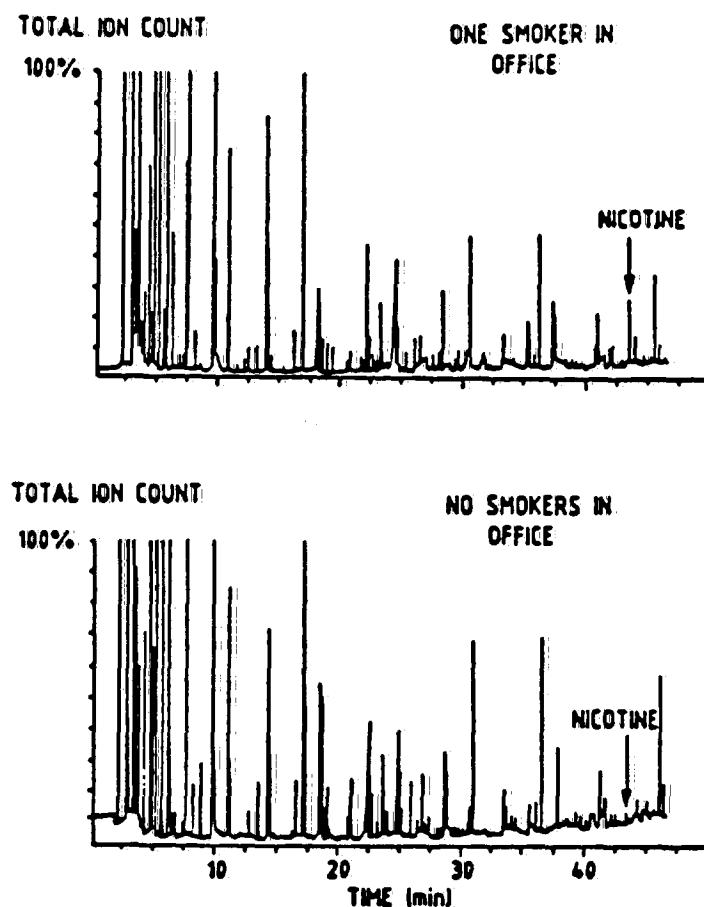


Fig. 7. Chromatographic profiles of the air in a smokers' and a nonsmokers' office in a modern, air-conditioned building.

tinguish between the VOC levels in smokers' and nonsmokers' offices (Bayer and Black 1987; Proctor et al. 1989b).

Fig. 7 illustrates a comparison of volatile chemicals in the air of a smoker's and a nonsmokers' office in the same building. The chromatographic profiles were acquired by drawing air through a sampling tube containing Tenax TA adsorbent for an hour sampling period. VOCs are trapped on the Tenax, and recovered for analysis by subsequent thermal desorption — capillary gas chromatography — mass spectrometry (Proctor et al. 1988b). The two offices were virtually identical, apart from the presence of one smoker (who smoked 3 cigarettes during the sampling period), and three nonsmokers in what is termed the smoker's office, and the presence of four nonsmokers in the other office. The nicotine peak in the smoker's office corresponded to a level of  $6 \mu\text{g m}^{-3}$ . Apart from nicotine, a detailed analysis, using mass

spectrometry revealed a similar range of volatile organic compounds (such as benzene and styrene) and similar concentrations of these chemicals in both the smoker's and nonsmokers' office. This finding is not surprising, as the nicotine peak would have to dominate the chromatographic profile in order that the VOC contribution from ETS could be detected above the background level (nicotine being the predominant ETS-associated volatile). A small amount of nicotine ( $0.5 \mu\text{g m}^{-3}$ ) was found in the nonsmokers' office, but the similarity in VOC levels between smoking and nonsmoking situations is not due to recirculation of chemicals from smoker's offices because both UV-RSP and nicotine levels in the nonsmoking areas were far lower (by a factor of at least 10) than in smoking areas.

Hence, the accurate assessment of ETS in real-life situations relies upon the use of specific chemical markers to distinguish the ETS contribution from the

2023380147

chemical background, arising from various sources, that is present whether smoking is taking place or not.

## REFERENCES

- Ayer, H.E.; Yeager, D.W. Irritants in cigarette smoke plumes. *Am. J. Pub. Health* 72:1283-1285; 1982.
- Baker, R.R. Product formation mechanisms inside a burning cigarette. *Prog. Energy and Comb. Sci.* 7:135-153; 1981.
- Baker, R.R. Variation of sidestream gas formation during the smoking cycle. *Beitr. Tabakforsch. Int.* 11:181-193; 1982.
- Baker, R.R.; Case, P.D.; Warren, N.D. The build-up and decay of ETS constituents as a function of room conditions. In: Parry, R.; Kirk, P.W., eds. *Indoor air and ambient air quality*. London: Selper; 1988:121-130.
- Bayer, C.W.; Black, M.S. Capillary chromatographic analysis of volatile organic compounds in the indoor environment. *J. Chrom. Sci.* 25:60-64; 1987.
- Black, A.; Pritchard, J.N.; Walsh, M. An exposure system to assess the human uptake of airborne pollutants by radio-tracer techniques. *J. Aerosol Sci.* 18:757-760; 1987.
- Blake, C.J.; Piadé, J.J.; Fink, W. Quantitative evaluation of sidestream smoke components under controlled experimental conditions. Paper presented at International experimental toxicology symposium on passive smoking. Essen: Institute of Hygiene and Occupational Medicine, Essen University; 1986.
- Browne, C.L.; Keith, C.H.; Allen, R.E. The effect of filter ventilation on the yield and composition of mainstream and sidestream smoke. *Beitr. Tabakforsch. Int.* 10:81-90; 1980.
- Brunnemann, K.D.; Hoffmann, D. The pH of tobacco smoke. *Food Control Technol.* 12:115-124; 1974.
- Cain, W.S.; Leederer, B.P. Odour control through ventilation: smoking vs non-smoking occupancy. Proc. 74th annual meeting of the Air Pollution Control Association. Vol. 2. Paper 81-22.5; 1981.
- Carson, J.R.; Erikson, C.A. Results from a survey of environmental tobacco smoke in offices in Ottawa, Ontario. *Environ. Tech. Lett.* 9:501-508; 1988.
- Case, P.D. The build-up and decay of ambient cigarette smoke components. Presented at the 39th tobacco chemists' research conference. Montreal: Canadian Tobacco Manufacturer's Council; 1985.
- Creighton, D.B. Tobacco smoke retention. Presented at Conference on aerosol physics. BAT, Southampton: British-American Tobacco Company; 1973.
- Eastough, D.J.; Benner, C.; Mooney, R.L.; Bartholomew, D.; Steiner, D.S.; Hanson, L.D.; Lamb, J.D.; Lewis, E.A.; Eastough, N.L. Gas and particle phase nicotine in environmental tobacco smoke. Proc. 79th annual meeting of the Air Pollution Control Association. Minneapolis. Vol. 5. Paper 86-68.5; Pittsburgh, PA: Air and Water Management Association; 1986.
- Eastough, D.J.; Benner, C.L.; Bayona, J.M.; Caka, P.M.; Mooney, R.L.; Lamb, J.D.; Lee, M.L.; Lewis, E.A.; Hansen, L.D.; Eastough, N.L. Identification of conservative tracers of environmental tobacco smoke. Proc. 4th international conference on indoor air quality and climate. Vol. 2. Seifert, B.; Radon, H.; Fischer, M.; Raden, H.; Wegner, J., eds. West Berlin: Institute for Water, Soil and Air Hygiene; 1987:1-7.
- Eastough, D.J.; Benner, C.L.; Bayona, J.M.; Richards, G.; Lamb, J.D.; Lee, M.L.; Lewis, E.A.; Hansen, L.D. Chemical composition of environmental tobacco smoke. I. Gas-phase acids and bases. *Environ. Sci. Technol.* 23:679-687; 1989.
- Eudy, L.W.; Thorne, F.A.; Heavner, D.L.; Green, C.R.; Ingebrethsen, B.J. Studies on the vapour-particle phase distribution of environmental nicotine by selective trapping and detection methods. Proc. 79th annual meeting of the Air Pollution Control Association. Vol. 2. Minneapolis. Paper 86-38.7; 1986.
- Girman, J.; Traynor, G. Indoor concentrations. *J. Am. Pollut. Contr. Assoc.* 33:89-95; 1983.
- Guerin, M.R. Formation and physico-chemical nature of sidestream smoke. In: O'Neill, I.K.; Brunnenmann, K.D.; Dodet, B.; Hoffmann, D., eds. *Environmental carcinogens: methods of analysis and exposure measurement*, Vol. 9: passive smoking. IARC Publication No. 81. Lyon, France; 1987.
- Halsey, N.J.; Sepkovic, D.W.; Brunnenmann, K.D.; Hoffmann, D. Biomarkers for assessing environmental tobacco smoke uptake. Proc. 81st annual meeting of the Air Pollution Control Association. Niagara Falls, NY; 1988.
- Hammond, S.K.; Leederer, B.P.; Soche, A.C.; Scheeler, M. Collection and analysis of nicotine as a marker for environmental tobacco smoke. *Atmos. Environ.* 21:457-462; 1987.
- Heavner, D.L.; Thorne, F.A.; Eudy, L.W.; Ingebrethsen, B.J.; Green, C.R. A test chamber and instrumentation for the analysis of selected environmental tobacco smoke (ETS) components. Proc. 79th annual meeting of the Air Pollution Control Association. Minneapolis. Vol. 2. Paper 86-37.9. Pittsburgh, PA: Air and Waste Association; 1986.
- Hiller, F.C.; Anderson, P.J.; Mazumder, M.K. Deposition of sidestream cigarette smoke in the human respiratory tract. *Toxicol. Letters* 35:95-99; 1987.
- Hoegg, U.R. Cigarette smoke in closed spaces. *Environ. Health Perspect.* 2:117-128; 1972.
- ISCSH (Independent Scientific Committee on Smoking and Health). Fourth Report. London: Her Majesty's Stationery Office; 1981.
- Ingebrethsen, B.J.; Sears, S.B. Particle size distribution measurements of sidestream cigarette smoke. Paper presented at the 39th tobacco chemists' research conference. Montreal: Canadian Tobacco Manufacturer's Council; 1985.
- Ingebrethsen, B.J.; Heavner, D.L.; Angel, A.L.; Conner, J.M.; Oldaker, G.B. III; Green, C.R. A comparative study of environmental tobacco smoke particulate mass measurements in an environmental chamber. Paper presented at the International experimental symposium on passive smoking. Essen: Institute of Hygiene and Occupational Medicine; 1986.
- Jenkins, R.A.; Guerin, M.R. Analytical chemical methods for the detection of environmental tobacco smoke constituents. *Eur. J. Respir. Dis. Suppl.* 133:33-46; 1984.
- Johanson, W.R.; Powell, D.H.; Hale, R.W.; Kornfeld, R.A. Incorporation of atmospheric oxygen into components of cigarette smoke. *Chem. Ind. (London)*, 521-522; 1975.
- Klus, H.; Kuhn, H. Distribution of various tobacco smoke components between mainstream and sidestream smoke (a survey). *Beitr. Tabakforsch. Int.* 11:229-265; 1982.
- Klus, H.; Beguer, H.; Nowak, A.; Seitz, M.; Utach, I. Measurements of ETS components in indoor air under controlled conditions. Presented at International conference on indoor air quality. Tokyo: The Council for Environment and Health; 1987.
- Lipp, G. On the definition of selectivity and the various smoke streams of the cigarette. *Beitr. Tabakforsch. Int.* 3:220-222; 1965.
- Manuel, N. Lung cancer and passive smoking. *Br. Med. J.* 294(6569): 440-441; 1987.
- McRae, D.D.; Jenkins, R.W., Jr. Use of a Schlieren optical system to observe the thermal column rising from a cigarette coal. Paper presented at the 41st tobacco chemists' research conference. Greensboro, NC: University of North Carolina; 1987.
- NRC (National Research Council). *Indoor air pollutants*. Washington, D.C.: National Academy Press; 1981.
- NRC (National Research Council). Committee on Passive Smoking. Board of Environmental Studies and Toxicology.

2023380148

- Environmental tobacco smoke — measuring exposures and assessing health risks. Washington, D.C.: National Academy Press; 1986.
- Neurath, G.; Ehmke, H.; Schneemann, H. On the water content of mainstream and sidestream smoke. *Beitr. Tabakforsch.* 3:351-357; 1966.
- Norman, V.; Ihrig, A.M.; Larson, T.M.; Moss, B.L. The effect of some nitrogenous blend components on NO/NO<sub>x</sub> and HCN levels in mainstream and sidestream smoke. *Beitr. Tabakforsch. Int.* 12:55-62; 1983.
- Nystrom, C.W.; Green, C.R. Assessing the impact of environmental tobacco smoke on indoor air quality. In: Proc. ASHRAE conference IAQ86. Managing indoor air for health and energy conservation. Atlanta, GA: American Society of Heating, Refrigerating and Airconditioning Engineers; 1986: 213-233.
- Ogden, M.W.; Maiolo, K.C. Gas chromatographic determination of solanesol in ETS. *J. High Resolut. Chromatogr. Chromatogr. Commun.* 11(4):341-343; 1988.
- Okada, T.; Ishizu, Y.; Matsunuma, K. Determination of particle-size distribution and concentration of cigarette smoke by a light scattering method. *Beitr. Tabakforsch.* 9:153-160; 1977.
- Olander, L.; Johansson, J.; Johansson, R. Air cleaners for tobacco smoke. Proc. 4th international conference on indoor air quality and climate. West Berlin. Seifert, B.; Esdorn, H.; Fischer, M.; Rüden, H.; Wegner, J., eds. Institute for Water, Soil and Air Hygiene, Berlin. Vol. 2.; 1987: 39-43.
- Oldaker, G.B. III; Coers, F.C. Estimation of the effect of ETS on air quality within passenger cabins of commercial aircraft. *Environ. Sci. Technol.* 1(10):994-999; 1987.
- Piatti, J.J.; Fink, W. Assessment of ETS impact to office air quality. Presented at International conference on indoor air quality. Tokyo: The Council for Environment and Health; 1987.
- Pritchard, J.N.; Black, A.; McAughey, J.J. The physical behaviour of sidestream tobacco smoke under ambient conditions. In: Indoor and ambient air quality. Perry, R.; Kirk, P.W., eds. London: Selper; 1988: 49-56.
- Proctor, C.J.; Martin, C.; Shillabeer, P.K.; Bevan, J.L.; Dymond, H.F. The evaluation of an apparatus designed for the collection of sidestream tobacco smoke. *The Analyst.* 113:1509-1513; 1988a.
- Proctor, C.J. The analysis of the contribution of ETS to indoor air. *Environ. Tech. Lett.* 9:553-562; 1988b.
- Proctor, C.J.; Warren, N.D.; Bevan, M.A.J. An investigation of the contribution of environmental tobacco smoke to the air in betting shops. *Environ. Tech. Lett.* 10:333-338; 1989a.
- Proctor, C.J. A comparison of the volatile organic compounds present in the air of real-world environments with and without environmental tobacco smoke. 82nd Annual meeting of the Air and Waste Management Association. Anaheim, California; paper 89-80.4. Pittsburgh, PA: Air and Waste Management Association; 1989b.
- Rawbone, R.G.; Burns, W.; Haslett, G. The ageing of sidestream tobacco smoke components in ambient environments. Paper presented at International conference on indoor air quality. Tokyo: The Council for Environment and Health; 1987a.
- Rawbone, R.G.; Burns, W.; Patrick, R.A. The measurement of 'environmental tobacco smoke' particulates. *Toxicol. Lett.* 35:125-129; 1987b.
- Repace, J.L. Indoor concentrations of environmental tobacco smoke: models dealing with effects of ventilation and room size. In: O'Neill, J.K.; Brunnenmaier, K.D.; Dodes, B.; Hoffman, D., eds. Environmental carcinogens, methods of analysis and exposure measurement. Vol. 9: passive smoking. Chapter 3. IARC Publication No. 81. Lyon, France; 1987a.
- Repace, J.L. Indoor concentrations of environmental tobacco smoke, field studies. In: O'Neill, J.K.; Brunnenmaier, K.D.; Dodes, B.; Hoffman, D., eds. Environmental carcinogens, methods of analysis and exposure measurement. Vol. 9: passive smoking. Chapter 10; p.25-41. IARC Publication No. 81. Lyon, France; 1987b.
- Repace, J.L.; Lowrey, A.H. A quantitative estimate of non-smokers' lung cancer risk from passive smoking. *Environ. Int.* 11:3-22; 1985.
- Robinson, D.P. Aerodynamic characteristics of the plume generated by a burning cigarette. Proc. International conference on the physical and chemical processes occurring in a burning cigarette. Wake Forest University, Winston-Salem, NC: R.J. Reynolds Tobacco Company; 1987: 115-150.
- Robinson, D.P.; Warren, N.D. Prediction of the build-up and decay of ETS components by mathematical modelling. In: Perry, R.; Kirk, P.W., eds. Indoor air and ambient air quality. London: Selper; 1988: 67-76.
- Sakuma, H.; Kusama, M.; Munakata, S.; Ohsumi, T.; Sugawara, S. The distribution of cigarette smoke components between mainstream and sidestream smoke. 1. acidic components. *Beitr. Tabakforsch. Int.* 12:63-71; 1983.
- Sakuma, H.; Kusama, M.; Yamaguchi, K.; Makuki, T.; Sugawa, S. The distribution of cigarette smoke components between mainstream and sidestream smoke. 2. bases. *Beitr. Tabakforsch. Int.* 12:199-209; 1984a.
- Sakuma, H.; Kusama, M.; Yamaguchi, K.; Sugawa, S. The distribution of cigarette smoke components between mainstream and sidestream smoke. 3. middle and higher boiling components. *Beitr. Tabakforsch. Int.* 12:251-258; 1984b.
- Samet, J.M.; Marbury, M.C.; Spengler, J.D. Health effects and sources of indoor air pollution, part I. *Am. Rev. Respir. Dis.* 136:1486-1508; 1987.
- Spengler, J.D.; Dockery, D.W.; Turner, W.A.; Wolfson, J.M.; Ferris, B.G. Jr. Long-term measurements of respirable sulphates and particulates inside and outside homes. *Atmos. Environ.* 13:23-30; 1981.
- Sterling, T.D.; Dimich, H. Indoor by-product levels of tobacco smoke. *J. Air Pollut. Contr. Assoc.* 32(3):250-259; 1982.
- Thomas, C.E.; Parmish, M.E.; Baker, P.G.; Fenner, R.A.; Tindall, J.E. The reproducibility of ETS measurements at a single site. 82nd annual meeting of the Air and Waste Management Association. , Anaheim, CA; paper 89-84.5. Pittsburgh, PA: Air and Waste Management Association; 1989.
- Thompson, C.V.; Jenkins, R.A.; Higgins, C.E. A thermal desorption method for the determination of nicotine in indoor environments. *Environ. Sci. Technol.* 23:429-435; 1989.
- Uberla, K. Lung cancer from passive smoking: hypothesis or convincing evidence. *Int. Arch. Occup. Environ. Health.* 69(5):21-37; 1987.
- USSG (U.S. Surgeon General) The health consequences of involuntary smoking; report of the U.S. Surgeon General. Washington, D.C.: U.S. Department of Health and Human Services; 1986.
- Umemura, S.; Muramatsu, M.; Okada, T. A study on precursors of nitric oxide in sidestream smoke. *Beitr. Tabakforsch. Int.* 13:183-190; 1986.
- Vu Duc, T.; C.K. Huynh Deposition rates of sidestream tobacco smoke particles and associated polycyclic aromatic hydrocarbons (PAHs). *Toxicol. Lett.* 35:59-65; 1987.
- Wallace, L.A.; Pallizzari, E.D.; Hartwell, J.D.; Spartacino, C.; Whitemore, R.; Sheldon, L.; Zelen, H.; Perritt, R. The TEAM study. *Environ. Res.* 43:290-307; 1987.

2023380149

## Characterization of Environmental Tobacco Smoke

Göran Löfroth,<sup>1</sup> Robert M. Burton,<sup>2</sup> Linda Forehand,<sup>3</sup> S. Katherine Hammond,<sup>1</sup> Robert L. Sells,<sup>2</sup> Roy B. Zweldinger,<sup>2</sup> and Joellen Lewtas<sup>1,2</sup>

Health Effects Research Laboratory and Atmospheric Research and Exposure Assessment Laboratory, U.S. Environmental Protection Agency, Research Triangle Park, North Carolina 27711; Environmental Health Research & Testing Inc., Research Triangle Park, North Carolina, and Department of Family and Community Medicine, University of Massachusetts Medical School, Worcester, Massachusetts

**NOTICE**  
This material may be protected by copyright law (Title 17 U.S. Code).

■ Environmental tobacco smoke (ETS) has been analyzed with respect to several components following smoking of research cigarettes in an experimental chamber. Parameters analyzed and their airborne yield per cigarette included particulate matter (10 mg) and its mutagenic activity in a *Salmonella* bioassay, carbon monoxide (67 mg), nitrogen oxides (2 mg), nicotine (0.8–3.3 mg), formaldehyde (2 mg), acetaldehyde (2.4 mg), crocetin (0.56 mg), benzene (0.5 mg), and several unsaturated aliphatic hydrocarbons (e.g., 1,3-butadiene) of which isoprene (3.1 mg) had the highest yield. ETS from commercial cigarettes was likewise analyzed in the experimental chamber and at a public location. The relative component composition for ETS is similar when generated from either research or commercial cigarettes. All components analyzed were present at concentrations above the background concentrations. Isoprene might be utilized as a tobacco smoke tracer for unsaturated aliphatic hydrocarbons.

### Introduction

Environmental tobacco smoke (ETS), which is derived primarily from sidestream smoke emitted between puffs, is a major contributor to indoor air pollution wherever smoking occurs (1, 2). ETS differs both chemically and physically from the precursor sidestream smoke, presumably due to chemical and physical transformations that occur as the mixture is diluted and aged. Chemical characterization studies have focused on mainstream and sidestream smoke (1). Data are lacking, however, on the presence and concentration of potentially toxic and carcinogenic components in tobacco-smoke-polluted indoor environments. An ideal ETS tracer air contaminant is not available for total ETS exposure (2), although nicotine is the best available tracer.

In this study we investigated the concentration of a number of genotoxic components as well as potential tracers of ETS under controlled and environmental conditions. Some of the components measured are routinely monitored air pollutants including carbon monoxide, nitrogen oxides, and particulate matter. A series of aldehydes and alkenes were measured in these studies, including several that are carcinogenic. The mutagenicity of the particulate phase was assayed in *Salmonella typhimurium*. Nicotine was measured as an ETS tracer. Indoor chamber experiments were performed at the EPA facility at the University of North Carolina, Chapel Hill, partly in conjunction with studies on the urinary cotinine (nicotine metabolite) concentration and excretion rate in young children following exposure to sidestream cigarette smoke.

(3). Indoor measurements were also made in a tavern.

### Experimental Section

**Chamber and Smoking.** The tests were performed in a 13.6-m<sup>3</sup> Plexiglas chamber (4) set at a ventilation rate of 3.55 air changes h<sup>-1</sup>; in addition, air removed by the sampling added ~0.50 air changes h<sup>-1</sup>. The air in the chamber was circulated by a fan at 1.35 m<sup>3</sup> h<sup>-1</sup>. The temperature and the relative humidity are given in Table I.

Research cigarettes of the type 2R1 (5), which had been equilibrated at 22 °C at 60% relativity humidity for 48 h, were smoked by machine (RM30, Heinr. Borgwalt, Hamburg, FRG). One cigarette was lighted every 30 min and was smoked with a 35-mL puff of 2 s every minute until extinguished after ~12 min. Mainstream smoke was vented to the outside of the chamber. The cigarettes weighed ~1.2 g, of which 0.9–1.0 g was consumed.

One adult and one child were present in the chamber during the 4-h tests in the first series of nine experiments. Six additional experiments were performed with the research cigarettes smoked by machine later in a second series, including two tests with no smoking, two tests (13 and 14) similar to the first series, (one cigarette every 30 min), and two tests (15 and 16) with one cigarette every 15 min. In tests 15 and 16, decay of components in the chamber was measured. Subsequently, in a third series of chamber tests, the emissions from two different commercial cigarette brands (A and B, both low-tar and -nicotine brands) were analyzed in the chamber with regular smoking by one person without any applied ventilation.

**Sampling and Analysis.** Particle Sampling and Analysis. Total suspended particles (TSP) were collected in duplicate on preweighed Teflon-coated glass fiber filters (Pallflex) at 1.7 m<sup>3</sup> h<sup>-1</sup> by modified Anderson samplers consisting of the 10-mm preseparator and the backup filter. TSP was also measured continuously by an Electric Aerosol Analyzer, EAA (Thermo-System, Inc., Model 3030), with measurements taken every 9 or 10 min. Particles were also collected in triplicate with personal sampling pumps (Model P4000, Du Pont, Kennett Square, PA) at 1.7 and 3 L min<sup>-1</sup>.

Nicotine. Nicotine was collected on bisulfate-impregnated filters (6) placed downstream from the particle filters on the personal samplers (first series) or on both Anderson and personal samplers (second series). Extraction and gas chromatography analysis of nicotine was performed as described by Hammond et al. (6).

Particle Mutagenicity. The filters were extracted by sonication in dichloromethane, and the extract was brought to a fixed volume. Aliquots of the solution were distributed into 4-mL vials together with 5 µL of dimethyl sulfoxide (DMSO) and then evaporated by nitrogen gas at 35 °C. The vials were kept capped at -20 °C until bioassayed.

The mutagenicity was determined by a microsuspension assay developed by Kado et al. (7) and modified by DeMarini et al. (in preparation) using *Salmonella* TA98 in the presence of S9 (8). The microsuspension was modified by using a bacterial suspension concentrated 5 times in-

<sup>1</sup>Visiting Scientist at U.S. EPA from Nordic School of Public Health, P.O. Box 12133, S-402 42 Gothenburg, Sweden.

<sup>2</sup>Atmospheric Research and Exposure Assessment Laboratory, U.S. EPA.

<sup>3</sup>Environmental Health Research & Testing Inc.

<sup>4</sup>University of Massachusetts.

<sup>5</sup>Health Effects Research Laboratory, Genetic Bioassay Branch, MD68, U.S. EPA.

**Table I.** Average Chamber Concentrations ( $\pm$ SD) and Average Airborne Yields for Carbon Monoxide, Nitrogen Oxides, Total Suspended Particles, Particle Mutagenicity, and Nicotine during Smoking of One Cigarette Every 30 Minutes (Tests 1-9, 12, and 14) and Every 15 Minutes (Tests 15 and 16) in a 18.6-m<sup>3</sup> Chamber with an Air Exchange of 3.55 h<sup>-1</sup>.

component	series I 1-9	series II 13 and 14	15 and 16	airborne yield per cigarette <sup>a</sup>
relative humidity, %	53 $\pm$ 4	29 $\pm$ 1	34 $\pm$ 0	b
temperature, °C	24 $\pm$ 0.5	23 $\pm$ 0	23 $\pm$ 0	b
carbon monoxide, mg/m <sup>3</sup>	2.45 $\pm$ 0.29	1.79 $\pm$ 0.81	4.76 $\pm$ 0.21	67 mg
nitric oxide, $\mu$ g/m <sup>3</sup>	68.0 $\pm$ 6.0 <sup>c</sup>	61.0 $\pm$ 2.8	139 $\pm$ 6	b
nitrogen oxides, <sup>c</sup> $\mu$ g/m <sup>3</sup>	72.8 $\pm$ 6.9 <sup>c</sup>	66.0 $\pm$ 8.5	139 $\pm$ 9	1960 mg
total suspended particles, $\mu$ g/m <sup>3</sup>				
by mass	349 $\pm$ 39	321 $\pm$ 25	934 $\pm$ 46	10 mg
by EAA <sup>d</sup>	349 $\pm$ 45 <sup>d</sup>	513 $\pm$ 47	1223 $\pm$ 84	b
mutagenicity, revertants/m <sup>3</sup>				
personal sampler	628 $\pm$ 49	nd <sup>e</sup>	nd	17300 revertants
Anderson sampler	494 $\pm$ 58	517 $\pm$ 40	837 $\pm$ 76	13400 revertants
nicotine, $\mu$ g/m <sup>3</sup>				
personal sampler 1-9	29 $\pm$ 7	nd	nd	800 mg
personal sampler 13-16	nd	127 $\pm$ 23	228 $\pm$ 49	3300 mg
Anderson sampler 13, 14	nd	160 $\pm$ 14	nd	b

<sup>a</sup>Uncorrected for surface removal. <sup>b</sup>Not applicable. <sup>c</sup>Based on eight tests. <sup>d</sup>Expressed as NO from total concentration of NO and NO<sub>2</sub>. <sup>e</sup>Assuming unit density. <sup>f</sup>Based on four tests. <sup>g</sup>nd, not determined.

stead of 10 times, 0.015 M phosphate-buffered saline instead of 0.15 M, no shaking during the 90-min incubation of the vials at 37 °C, and addition of histidine and biotin to the plate bottom agar instead of to the top agar.

The combined sample from the duplicate Anderson filters from each experiment was tested with six doses corresponding to 25–300 L of air in duplicate tests with duplicate vials for each dose and test. The combined sample from the personal filters from each experiment was tested with three doses corresponding to 50–200 L of air in one test, with triplicate vials for each dose. The response was calculated by linear regression using doses on the linear or almost linear part of the dose-response curve.

**Carbon Monoxide and Nitrogen Oxides.** Carbon monoxide (CO) was measured continuously by nondispersive infrared absorption (Bendix 8501-5), and nitrogen oxides NO<sub>x</sub> (i.e., NO plus NO<sub>2</sub>) were measured indirectly by chemiluminescence (Bendix 8101-B). Data points were recorded every 3 min.

**Hydrocarbons.** Air was collected in evacuated stainless steel canisters (9), and the sample was then subjected to speciated gas chromatographic analysis by the method described by McElroy et al. (10). Samples in the first experimental series were collected as grab samples at a peak concentration of carbon monoxide in the chamber, whereas samples in the second series were collected over the entire smoking period (4 h).

**Aldehydes.** Aldehydes were collected in the second series at a rate of 1.0 L min<sup>-1</sup> using 2,4-dinitrophenyl-hydrazine-coated silica gel cartridges for collection and high-performance liquid chromatography for analysis of the hydrazone derivatives as described by Tejada (11).

**Calculations.** The average chamber concentrations were calculated as the average value between 1 h after start until the end of the experiments. When sampling included the first hour, the average concentration was calculated by normalizing to the continuous CO concentration; this correction was approximately 5%. Likewise, grab samples of hydrocarbons were normalized to the average concentration from the peak concentration, when the sample had been collected. The airborne yield, expressed as amount per cigarette, was calculated from the average concentration by using the known smoking frequency, the chamber volume, and the total air exchange rate.

**Environmental Sampling.** The impact of tobacco smoke was determined in two studies in a local tavern. The main room in which sampling took place had a volume

of  $\sim$ 180 m<sup>3</sup> ( $l = 15$  m,  $w = 4$  m, and  $h = 3$  m) and was variously occupied by 5–25 persons, many of whom were smoking.

Indoor TSP and nicotine were collected on a Teflon-coated glass fiber filter and a second bisulfate-impregnated filter, respectively, at 20 L/min by an Anderson sampler. Particulate matter was measured by taking 120-s readings each 1/2 h over the 3- or 4-h study with a piezobalance Model 3500 (TSI Inc., St. Paul, MN) both indoors and outdoors with at least two cleaning cycles per hour. Indoor and outdoor carbon monoxide was determined with two General Electric Model 15ECS3CO3 carbon monoxide detectors (Wilmington, MA) that had been calibrated at zero and 60 ppm CO. Indoor aldehydes and indoor and outdoor hydrocarbons were collected and analyzed as described for the chamber studies. The hydrocarbon sampling was performed during only 2 h in each of the two studies.

#### Results

The concentrations and calculated yields are given in Table I for components that were analyzed in all chamber tests in the first and second series. Carbon monoxide and nitrogen oxides were determined continuously every 3 min, and their concentrations varied in a saw-toothed form with the smoking cycle of one cigarette every 30 min. The ratio of the average maximum to the minimum concentration was  $\sim$ 3. The average concentration of carbon monoxide was about 65–70% of the maximum concentration; similar ratios were found for nitrogen oxides.

Particle concentrations measured by EAA had the same type of variation, but the resolution was less because the analyses were performed less frequently. The average concentration of particles as measured by EAA (assuming unit density) was in good agreement with the concentration obtained by filter collection in the first series and overestimated the particle concentration in the second series under lower relative humidity. Due to the organic character of ETS, however, the density would be expected to be somewhat less than 1.0.

The nicotine concentrations and yields were lower during the first series than during the second series, with yields of 800 mg/cigarette and 3300 mg/cigarette, respectively. There were several differences in the two series. In the first series, the chamber contained more adsorbant surfaces: two persons, mother and child, television set, crib, chair, and a curtain, all of which were absent in the

**Table II. Selected Hydrocarbon Concentrations ( $\mu\text{g}/\text{m}^3$ ) in Background Air and in the 14.4'-ft<sup>3</sup> Chamber with Sixtures**

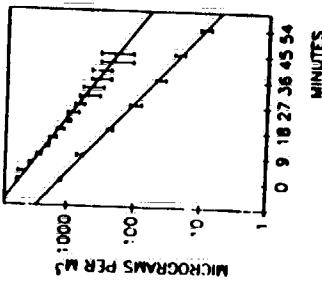
grab sample at peak concn	continuous sampling						av airborne yield, ug/cigarette						
	test	1	2	6	av	background	test	13	av concn*	background	test	15	av concn*
hydrocarbon	background												
ethane	65	116	106	136	35	42	92	50	67	116	98	56	1200
ethene	5	63	93	69	48	16	77	61	23	161	138	107	1600
propene	2	74	77	64	45	3	48	45	2	107	106	106	1300
propane	9	54	56	50	29	12	39	27	15	84	69	69	800
1,3-butadiene	<1	22	24	18	14	0.6	11	12	0.6	20	19	400	
isoprene	11	189	231	128	123	2	94	92	8	203	225	3100	
benzene	3	35	38	29	20	4	19	15	3	40	37	500	

\* Above the background concentration. \* Uncorrected for surface removal. \* Estimated as ethene, as ethene and acrylene concentrations graphed.

**Table III. Selected Aldehyde Concentrations ( $\mu\text{g}/\text{m}^3$ ) in Background Air and in the 14.4'-ft<sup>3</sup> Chamber with Sidestream Cigarette Smoke, and Their Airborne Yields**

sample	formaldehyde		acetaldehyde	acrolein
test 11, no smoking	14	16	<0.2	
test 12, no smoking	14	12	<0.2	
test 13, 1 cigarette/30 min	80	87	18	
test 14, 1 cigarette/30 min	84	84	19	
test 15, 1 cigarette/15 min	170	206	49	
test 16, 1 cigarette/15 min	173	233	39	
av airborne yield,* ug/cigarette	2000	2400	560	

\* Uncorrected for surface removal.



**Figure 1. Decay of carbon monoxide (upper curve) and particle (lower curve) concentrations in the chamber after the end of smoking. The values represent the average of tests 15 and 16 with bars giving the highest and lowest values.**

second series. The relative humidity was about 50–60% during the first series but only about 30% in the second series. Although surface area is likely to alter surface deposition, relative humidity has also been shown to effect surface deposition (B. Lauderer, personal communication).

Hydrocarbon concentrations and calculated yields are given in Table II. There was a good agreement between the two different series, the first of which comprised three grab samples at peak concentration and the second with continuously collected samples. Among the hydrocarbons analyzed, ethene had an exceptionally high background, which has been previously observed (J. J. Bufalini and B. W. Gay, Jr., personal communication). The analyses of selected aldehydes are given in Table III together with the calculated yields. The four tests gave results in close agreement with each other.

The limited study of the decay of carbon monoxide and

particle measured by EAA that were performed in tests 15 and 16 (Figure 1) showed that these components had

a mean ventilation time of about 14 and 11 min, respectively.

The ventilation time of carbon monoxide corresponds to the predetermined air exchange rate of ~4 changes/h. The disappearance of particle is ~25% faster than the disappearance of carbon monoxide under the experimental conditions used.

EETs from two commercial brands of cigarettes was analyzed following smoking of 1 or 2 cigarettes by a smoker located in the chamber. Although the airborne yields cannot be calculated, as only peak concentrations were measured, the relative proportions of the components show that the composition of the smoke is similar to that from research cigarettes (Table IV).

**Table IV. Peak Concentrations of Five EETs Components from Smoking of Two Commercial Cigarette Brands and Comparison with the Average Chamber Concentrations with 2E1 Research Cigarettes**

component	cigarette		brand A (2)		brand B (1)		2E1 av chamber concen*
	2E1	outdoor	indoor	outdoor	indoor	outdoor	
carbon monoxide, $\mu\text{g}/\text{m}^3$	9.3	—	—	—	—	—	2.4
nitrogen dioxide, $\mu\text{g}/\text{m}^3$	200	150	150	71	71	71	—
particles by EAA, $\mu\text{g}/\text{m}^3$	—	—	—	—	—	—	0.44
ethene, $\mu\text{g}/\text{m}^3$	200	110	110	45	45	45	—
isoprene, $\mu\text{g}/\text{m}^3$	650	280	280	110	110	110	—

\* Recalculated from Tables I and II. \* Above the maximum measurable concentration of the EAA.

**Table V. Air Pollutant Concentrations Indoors in a Tobacco and Biomass-heated House in the Ambient Outdoor Air**

component	first study (870504) 3 h		second study (870505) 4 h	
	indoor	outdoor	indoor	outdoor
TSP by weight	470	—	—	—
by piezobalance	420	220	220	220
mutagenicity, revertants/m³	800	800	800	800
carbon monoxide, $\mu\text{g}/\text{m}^3$	71	24	44	44
ethane, $\mu\text{g}/\text{m}^3$	—	—	—	—
formaldehyde	104	28	28	28
acetaldehyde	182	28	28	28
acrolein	24	21	21	21
hydrocarbons, $\mu\text{g}/\text{m}^3$	—	—	—	—
ethene	110	12	12	12
ethane	160	9	88	8
propane	70	6	40	6
1,3-butadiene	19	7	32	7
isoprene	180	7	11	1
benzene	27	6	28	2

\* Ind. not determined. \* Only 2 h of sample.

The ventilation time of carbon monoxide corresponds to the predetermined air exchange rate of ~4 changes/h. The disappearance of particles is ~25% faster than the disappearance of carbon monoxide under the experimental conditions used.

EETs from two commercial brands of cigarettes was analyzed following smoking of 1 or 2 cigarettes by a smoker located in the chamber. Although the airborne yields cannot be calculated, as only peak concentrations were measured, the relative proportions of the components show that the composition of the smoke is similar to that from research cigarettes (Table IV).

Finally, available portable equipment was used to sample and analyze the air in a tavern during normal smoking conditions. All ETS components that were analyzed indoors and outdoors were highly elevated in the indoor environment (Table V). Furthermore, none of the analyzed components was conspicuously much higher or much lower relative to each other than what would have been expected from the studies of the airborne yield of research cigarettes.

#### Discussion

This study characterizes both exposure concentrations and airborne yields for particulate matter and its mutagenic activity, as well as nicotine, aldehydes, and alkenes. This study provides documentation that the chamber ETS exposure was comparable to that which people would encounter in indoor environments where tobacco is being smoked. Additional chemical analyses and subsequent studies were conducted to relate the chamber ETS components to the analysis of ETS in an indoor environment.

ETS and other air pollutants emitted into an indoor environment can disappear by three routes: ventilation, surface deposition, and chemical reactions while airborne. The ventilation rate in most indoor environments is 0.5 air change/h or higher, which means that the time frame of interest is a few hours or less. Whereas few, if any, studies have dealt with chemical reactions of ETS components, there is good evidence that smoke particles can be removed by surface deposition (2, 12), the process being dependent on surface characteristics and mixing ratio. Thus, the airborne yield of particulate tar can vary depending on the experimental conditions.

Among the gases studied, carbon monoxide is considered to be sufficiently stable to be removed only by ventilation. This is also probably the case for the low molecular weight hydrocarbons, ethene to isoprene, analyzed in the present investigation, whereas nicotine as well as the aldehydes may decay by surface adsorption or reaction. This phenomenon may account for the divergent nicotine concentrations found in the first and second series (Table I), because the chamber was different with respect to surface characteristics in these two series.

Nitric oxide (NO) is the primary nitrogen oxide formed in tobacco smoke (13), but it can slowly be oxidized to nitrogen dioxide, NO<sub>2</sub>, (14), or species detected as NO<sub>x</sub>. The low contribution of NO<sub>2</sub> to the total concentration of nitrogen oxides found in the present study (Table I) most likely reflects the high ventilation rate, which would not give sufficient time for the formation of NO<sub>2</sub> from NO in the chamber. In contrast, about 15–25% of the nitrogen oxides detected in the smoking of commercial cigarettes (Table IV) was in the form of NO<sub>2</sub>, indicating that both the higher concentration and the lower ventilation rate in these tests resulted in a significant conversion.

The determined ETS airborne yields of carbon monoxides, nitrogen oxides, and nicotine (Table I) are about the same as those reported for sidestream smoke from commercial cigarettes (13, 15, 16). The ETS yield of particles is, however, lower by a factor of 2–3 than those reported for sidestream smoke from commercial cigarettes (16). One study of 2R1 cigarettes by Ueno and Peters (17) found only 6–9 mg of particulate matter/cigarette based on a sample collected with an Anderson cascade impactor and 1–2 mg/cigarette based on EAA measurements.

The mutagenic yield of particulate matter, 13 400–17 300 revertants/cigarette, is lower than the mutagenic emission, 36 500–118 000 revertants/cigarette, for sidestream cigarette smoke collected in a small hood (18). These differences may be due to the differences between the sample

collection methods for sidestream smoke and ETS and between the emission rate and airborne yield measurements. In both cases, the loss of mutagens associated with particles is likely due to loss of the particles to surfaces.

The mutagenicity concentrations and yields determined on particle extracts from the personal sampler were consistently higher than those from the modified Anderson sampler. The personal samplers have a 2-fold lower face velocity ( $1.3 \text{ cm s}^{-1}$ ) compared to the Anderson sampler ( $2.7 \text{ cm s}^{-1}$ ). The higher face velocity of the Anderson sampler may result in the loss of the more volatile organics and mutagens from the filter. Recent studies comparing face velocities substantiate this hypothesis (K. Hammond et al., unpublished data). The mutagenic response of 400–900 revertants/m<sup>3</sup> of air is a range that may be encountered in moderately smoky environments and is higher than that found for ambient outdoor air (19).

Airborne yield is a direct measure of the components present in a particular indoor environment and will vary with the surface area and characteristics. The advantage of this measurement is that it can be directly used to estimate indoor ETS compound concentrations based on the number of cigarettes smoked and the ventilation. Emission factors for sidestream cigarette smoke have classically been determined by using a small-volume collecting device surrounding the cigarette tip (16), and emission factors for ETS particles and nicotine have been determined in chambers with correction of surface removal (6). Thus, it can be expected that airborne yields are less than emission factors for components that are significantly removed by processes other than ventilation.

The emission of aliphatic hydrocarbons in sidestream smoke has not been assessed quantitatively previously, although there are several earlier studies on mainstream smoke that have been summarized by Elmenhorst and Schultz (20). The airborne yields for sidestream smoke found in the present study (Table II) are generally higher than those found for mainstream smoke. Isoprene is the predominant unsaturated hydrocarbon in sidestream smoke (Tables II, IV, and V), and the concentrations measured were well above the background concentrations. There are several other sources for isoprene: It is exhaled by man (21, 22) and rodents (23) and thus possibly by other animals. It is emitted from vegetation, with ambient concentrations generally below 15 mg/m<sup>3</sup> (24, 25). It is also produced during combustion, but the most likely combustion source, wood combustion, gives much less isoprene than ethene (26), indicating that isoprene is a minor constituent of the hydrocarbon emission. The carbonyl compounds studied (Table III) had airborne yields of a magnitude reported earlier (15).

The results presented in Table IV, obtained in the smoking of commercial cigarettes in the chamber, show that there is a similar relative distribution of major components from such cigarettes when compared to the airborne yields from the research cigarettes 2R1. This is also the fact for components measured in a tavern (Table V) in which a mixture of commercial cigarettes was being smoked. These comparisons between research cigarettes and commercial cigarettes show that both types of cigarettes give rise to ETS with very similar composition.

The ratio of nicotine to particles is ~160 µg/mg for the tavern samples (Table V), which is intermediate between the ratios that can be calculated for the chamber study, 80 and 330 µg/mg. This may indicate that the tavern has surface characteristics with respect to nicotine removal intermediate between the occupied and unoccupied Plexiglas chamber.

2023380153

It is well-known that tobacco smoking causes cancer, and recently a series of epidemiological studies reviewed by the National Research Council (2) and others (27, 28) have reported excess lung cancer deaths in individuals exposed to ETS. Cancer from passive smoking at sites other than the lung is also a possibility (29).

It is not known which of the many components present in tobacco smoke and ETS are the most hazardous. It is therefore important to analyze ETS for a variety of components comprising both particulate matter and gas-phase constituents. We have in the present study determined particulate matter and used a *Salmonella* mutagenicity bioassay to measure genotoxicity (30). Among the numerous gas-phase compounds in ETS, aldehydes (7) and unsaturated aliphatic hydrocarbons (31) are known or potential animal carcinogens. Although these compounds have a relatively low carcinogenic potency, they might be of importance in the total evaluation because they are present in relatively high concentration. Among the unsaturated hydrocarbons, isoprene might be used as a tobacco smoke tracer considering the low background concentration of this compound. Studies are needed to examine other potential indoor sources of isoprene that could interfere with its use as a tracer.

#### Acknowledgments

We thank A. A. Strong for operation of the chamber, M. M. Dallas for technical bioassay support, and D. M. DeMarini for advice and review comments.

Registry No. Carbon monoxide, 630-08-0; nitric oxide, 10102-43-9; nitrogen oxides, 11104-93-1; nicotine, 54-11-6; ethene, 74-85-1; ethane, 74-84-0; propene, 115-07-1; propane, 74-98-6; 1,3-butadiene, 106-99-0; isoprene, 78-78-6; benzene, 71-43-2; formaldehyde, 50-00-0; acetaldehyde, 75-07-0; acrolein, 107-02-8.

#### Literature Cited

- (1) *Tobacco Smoking*, IARC Monographs on the Evaluation of the Carcinogenic Risk of Chemicals; International Agency for Research on Cancer: Lyon, France, 1986.
- (2) *Environmental Tobacco Smoke*, National Research Council; National Academy Press: Washington, DC, 1986.
- (3) Goldstein, G. M.; Collier, A.; Etzel, R.; Lewtas, J.; Haley, N. In *Indoor Air Quality and Climate*; Seifert, B. et al., Eds.; Institute for Water, Soil and Air Hygiene: Berlin, FRG, 1986; Vol. 2, pp 61-67.
- (4) Strong, A. A.; Penley, R.; Knauf, J. H. Human exposure system for controlled ozone atmosphere. U.S. Environmental Protection Agency, Research Triangle Park, NC, 1977; EPA-600/1-77-048.
- (5) *Tobacco and Health Research Institute The Reference and Research Cigarette Series*; University of Kentucky: Lexington, KY, 1984.
- (6) Hammond, S. K.; Leaderer, B. P.; Rocha, A. C.; Schenker, M. *Atmos. Environ.* 1987, 21, 457-462.
- (7) Kado, N. Y.; Langley, D.; Eisenstadt, E. *Mutat. Res.* 1983, 121, 25-32.
- (8) Ames, B. N.; McCann, J.; Yamasaki, E. *Mutat. Res.* 1975, 31, 347-364.
- (9) Oliver, K. D.; Phail, J. D.; McClenny, W. A. *Atmos. Environ.* 1984, 20, 1403-1411.
- (10) McElroy, F. F.; Thompson, V. L.; Holland, D. M.; Loman, W. A.; Seila, R. L. *J. Air Pollut. Control Assoc.* 1984, 36, 710-714.
- (11) Tejada, S. B. *Int. J. Environ. Anal. Chem.* 1986, 26, 187-188.
- (12) Repace, J. L.; Lowrey, A. H. *Science* 1986, 238, 464-472.
- (13) Norman, V.; Irig, A. M.; Larson, T. M.; Moss, B. L. *Tabaksforsch. Int.* 1983, 12, 55-62.
- (14) Vilcins, G.; Lephardt, J. O. *Chem. Ind. (London)* 1975, 974-975.
- (15) Klus, H.; Kuhn, H. *Beitr. Tabaksforsch. Int.* 1982, 11, 229-265.
- (16) Richert, W. S.; Robinson, J. C.; Collishaw, N. *Am. J. Public Health* 1984, 74, 228-231.
- (17) Ueno, Y.; Peters, L. K. *Aerosol Sci. Technol.* 1986, 5, 469-476.
- (18) Ling, P. L.; Löfroth, G.; Lewtas, J. *Toxicol. Lett.* 1987, 35, 147-151.
- (19) Löfroth, G.; Ling, P. L.; Agurell, E. *Mutat. Res.* 1983, 202, 103-110.
- (20) Elmenhorst, H.; Schulz, C. *Beitr. Tabaksforsch. Int.* 1983, 4, 90-123.
- (21) Conkle, J. P.; Camp, B. J.; Welch, B. E. *Arch. Environ. Health* 1976, 30, 290-295.
- (22) Gelmont, D.; Stein, R. A.; Mead, J. P. *Biochem. Biophys. Res. Commun.* 1981, 99, 1455-1460.
- (23) Peter, H.; Wiegand, H. J.; Bolt, H. M.; Greim, H.; Walter, G.; Berg, M.; Pilser, J. G. *Toxicol. Lett.* 1987, 36, 9-14.
- (24) Lamb, B.; Westberg, H.; Allwine, G. *Atmos. Environ.* 1986, 20, 1-8.
- (25) Trainer, M.; Williams, E. J.; Parriah, D. D.; Buhr, M. P.; Allwine, E. J.; Westberg, H. H.; Pehsenfeld, F. C.; Liu, S. C. *Nature* 1987, 329, 705-707.
- (26) Kleindienst, T. E.; Shepon, P. B.; Edney, E. O.; Claxton, L. D.; Cupitt, L. T. *Environ. Sci. Technol.* 1986, 20, 603-601.
- (27) Wald, N. J.; Nanchahal, K.; Thompson, S. G.; Cuckle, H. S. *Br. Med. J.* 1986, 293, 1217-1222.
- (28) Saracci, R.; Riboli, E. *Mutat. Res.* 1989, 222, 117-127.
- (29) Pershagen, G. *Mutat. Res.* 1989, 222, 129-135.
- (30) Claxton, L.; Morin, R. S.; Hughes, T. J.; Lewtas, J. *Mutat. Res.* 1981, 222, 81-93.
- (31) Löfroth, G. *Mutat. Res.* 1983, 222, 73-80.

Received for review May 11, 1989. Revised manuscript received November 4, 1989. Accepted December 15, 1989. The research described in this article has been reviewed by the Health Effects Research Laboratory, U.S. Environmental Protection Agency, and approved for publication. Approval does not signify that the contents necessarily reflect the views and policies of the Agency nor does mention of trade names or commercial products constitute endorsement or recommendation for use.

2023380154

NOTICE  
This material may be  
protected by copyright  
law (Title 17, U.S. Code).

## Importance of exposure to gaseous and particulate phase components of tobacco smoke in active and passive smokers

Gerhard Scherer<sup>1</sup>, Charlotte Conze<sup>1</sup>, Lutz v. Meyerinck<sup>1</sup>, Marja Sorsa<sup>2</sup>, and Franz Adlikofer<sup>1</sup>

<sup>1</sup>Analytisch-biologisches Forschungslabor Prof. Dr. F. Adlikofer, Goethestrasse 20, D-8000 München 2, Federal Republic of Germany

<sup>2</sup>Institute of Occupational Health Department of Industrial Hygiene and Toxicology, Topeliuksenkatu 41 a A, SF-00250 Helsinki, Finland

Received January 25 / Accepted July 24, 1990

**Summary.** The uptake of tobacco smoke constituents from gaseous and particulate phases of mainstream smoke (MS), inhaled by smokers, and of environmental tobacco smoke (ETS), breathed in by non-smokers, was investigated in two experimental studies. Tobacco smoke uptake was quantified by measuring carboxyhemoglobin (COHb), nicotine and cotinine in plasma and urine and the data obtained were correlated with urinary excretion of thioethers and of mutagenic activity. An increase in all biochemical parameters was observed in smokers inhaling the complete MS of 24 cigarettes during 8 h, whereas only an increase in COHb and, to a minor degree, in urinary thioethers was found after smoking the gas phase of MS under similar conditions. Exposure of non-smokers to the gaseous phase of ETS or to whole ETS at similar high concentrations for 8 h led to identical increases in COHb, plasma nicotine and cotinine as well as urinary excretion of nicotine and thioethers which were much lower than in smokers. Urinary mutagenicity was not found to be elevated under either ETS exposure condition. As shown by our results, the biomarkers most frequently used for uptake of tobacco smoke (nicotine and cotinine) indicate on the one hand the exposure to particulate phase constituents in smoking but on the other hand the exposure to gaseous phase constituents in passive smoking. Particle exposure during passive smoking seems to be low and a biomarker which indicates ETS particle exposure is as yet not available. These findings emphasize that risk extrapolations from active smoking to passive smoking which are based on cigarette equivalents or the use of one biomarker (e.g. cotinine) might be misleading.

**Key words:** Environmental tobacco smoke (ETS) – Passive smoking – Active smoking – Gasphase – Particulate phase

Offprint requests to: G. Scherer

### Introduction

Exposure to environmental tobacco smoke (ETS) has been associated with adverse effects on the health of non-smokers [38]. Extrapolations made from the lung cancer risk seen in smokers suggest a small excess risk in passive smokers compared to non-exposed persons. Russell [30], for example, compared the cotinine levels in body fluids of active smokers and non-smokers and estimated by linear extrapolation that passive smoking might account for 1000 excess deaths per year in the UK and 4000 in the USA. Repace and Lowrey [29], using an extrapolation based on "cigarette equivalents" from particle measurements in smoke-filled rooms arrived at a figure of 5000 excess lung cancer deaths each year in the USA from passive smoking. Doll [6], however, has criticized this approach, partly because it is uncertain whether a linear or quadratic relationship between dose and effect in active smokers should be postulated, but also because of the many physical and chemical differences which exist between MS and ETS.

In order to assess the potential differences in exposure to MS and ETS by active and passive smokers, we have conducted a series of experimental studies on smokers and non-smokers. In the first study, smokers smoked their customary cigarette, inhaling whole MS on one day. On another day, the same number of cigarettes were smoked through a filter which retained the particles from MS, exposing the smokers to only the gaseous phase of the smoke. In the second study, non-smokers were exposed to the gaseous phase of ETS on one day and whole ETS on another. The uptake of potentially genotoxic compounds under these different exposure conditions was determined by measuring urinary mutagen and thioether excretion and assessed by comparison with dosimetric measures of uptake of various tobacco smoke constituents.

2023380155

## Materials and methods

**Subjects.** For Study 1, eight healthy male smokers aged 20 to 40 took part in the investigation. They reported to the laboratory on a Sunday evening and remained there until the end of the study on Friday morning. In Study 2, five healthy male non-smokers aged 23 to 29 years remained under similar controlled conditions from a Saturday evening until the following Friday morning. All subjects completed a questionnaire on socio-economic and lifestyle factors as well as their smoking habits. ETS exposure and meals consumed during the preceding 48 h. During the study periods, smokers were not allowed to smoke except during the special smoking sessions. During ETS exposure (Study 2), the smoke was generated by five smokers who were not allowed to smoke except during the smoking periods. During both studies, subjects ate a defined diet low in polycyclic aromatic hydrocarbons [23] which was identical in quality and quantity on each day of the investigation.

**Protocol of Study 1 (active smoking).** During the smoking days of the study, the eight active smokers smoked 24 cigarettes/d of their usual brand. The average tar and nicotine yields of these products were 13.1 and 0.95 mg, respectively. The protocol began with a control day (Day 1) during which smoking was not allowed. On Day 2 (Tuesday), the subjects smoked 24 cigarettes between 8:30 and 17:00 h (1 cig/20 min). To obtain conditions similar to those to be applied on Day 4, an empty filterholder was placed between the cigarette and the mouthpiece. Wednesday (Day 3) was a control day without smoking and on Day 4 (Thursday) the subjects smoked 24 cigarettes with a filter placed in the filterholder between the cigarette and the mouthpiece. The filters were of two types. Subjects 1 to 4 used a glass fibre filter (Millipore SJA V 013 NS, Schleicher & Schüll, FRG), while Subjects 5 to 8 used a Cambridge filter. Both filter systems had 99% effectiveness in removing smoke particles and were changed after every two cigarettes. Analysis revealed no difference in the particle removal by the two filters and the data obtained from Subjects 1 to 8 have therefore been pooled for analysis. The room used for the smoking study was adequately ventilated and ETS exposure of the smokers was minimal.

**Protocol of Study 2 (passive smoking).** On the first day of the study (Sunday) the subjects were neither exposed to ETS nor sham-exposed. The smokers were not allowed to smoke. On the second day of the study (Monday) the subjects spent 8 h (8:30 to 16:30 h) in an unventilated, furnished room (45 m<sup>2</sup>) in order to simulate exposure conditions (sham exposure). The third day (Tuesday) was an exposure day on which the five non-smokers were exposed to the gaseous phase of ETS during 8 h (8:30 to 16:30 h). The experimental procedure for this day was as follows: The five non-smokers sat in the experimental room wearing masks equipped with filters (Sekur Polimask-PC, filter classes P1 and P2, Pirelli, FRG) which retain more than 99% of the particle mass in the inspired air. The fourth day of the study (Wednesday) was a sham exposure day identical to Day 2. On the fifth day (Thursday) the non-smokers were exposed to whole ETS. The smokers generating ETS smoked the same numbers of cigarettes according to the same time schedule as on Day 3. The analysis outlined in the present paper is based only on data of passive smokers.

**Air analysis.** The air sampling tubes were installed at breathing height of a seated person at the end of the room opposite to where the smokers sat. Carbon monoxide was measured continuously by an infrared CO monitor UNOR 6N (Fa. Maihak, Hamburg, FRG). Nitrogen oxides (NO/NO<sub>2</sub>) were detected by a chemoluminescence monitor using a Nitrogen-Oxide Analyzer, Model 8840 (Monitor Labs Inc., USA). Nicotine was determined according to the method of Odgen [26]. The alkaloid was absorbed on XAD-4 resin with an air flow rate of 1 litre/min. Sampling times were 4 h on the sham-exposure days (Days 2 and 4) and 2 h on the exposure days (Days 3 and 5). Formaldehyde was absorbed on Sep-PAK C<sub>18</sub> (Waters Associates, Milford, MA, USA) coated with 2,4-dinitrophenylhydrazine and determined by HPLC [20]. Flow rates and

sampling times were as those for nicotine. Respirable particles were determined gravimetrically according to the method of Connor [5]. The sampling flow rate was 1.6 l/min. Sampling periods were similar to those for nicotine. Polycyclic aromatic hydrocarbons (PAH) were detected according to the method of Grimmer et al. [8]. Sampling period was 8 h. The filter system consisted of a siliconized glass fibre filter and a Pora Pak PS filter for sampling particles and semi-volatiles, respectively.

**Biomonitoring.** Urine was collected from all subjects at 24-h intervals beginning at 8:00 h on each day. Blood was drawn into heparinized tubes at 8:00 and 17:00 h from each subject, with an additional blood sample drawn from the active smokers (Study 1) on the two smoking days at 12:30 h after 12 cigarettes had been smoked. In Study 1, exhaled air was also sampled into plastic bags [10] on an hourly basis from 8:30 to 17:30 h, using a controlled collection procedure. Carbon monoxide in exhaled air was determined by an UNOR 6N CO monitor (Maihak, Hamburg, FRG). Carboxyhemoglobin (COHb) was determined spectrophotometrically on fresh blood samples with an IL 182 CO-Oximeter (IL 182, Instrumentation Laboratories Ltd., USA). Nicotine was determined in plasma and urine by gas chromatography [12] and cotinine in plasma by radioimmunoassay [21]. Thioethers were measured in urine by quantifying the sulphydryl groups with Ellman's reagent after alkaline hydrolysis of the thioether bonds according to the method of Heinonen et al. [11] as modified according to Aringer and Lidums [1]. Briefly, 5 ml of urine were extracted three times with 8 ml of ethylacetate. The pooled extracts were evaporated to dryness. The residue was dissolved in 2 ml of 2 M ascorbic acid. One ml of this solution was hydrolysed with 0.5 ml 4 N NaOH in the dark under nitrogen for 60 min at 100°C. The SH-groups in both the unhydrolysed and hydrolysed extracts were determined with Ellman's reagent (0.4 g 5,5-dithiobis-(2-nitrobenzoic acid) (DTNB) in 1 l of 1% citrate solution) at 412 nm. Parallel thioether analyses were performed in another laboratory. Results obtained by the two laboratories were not completely concordant and reasons for the discrepancy are under investigation. Analysis of either thioether data set are, however, consistent with the conclusions of this paper.

Urinary mutagenicity was determined as previously described using the *Salmonella typhimurium*/mammalian microsome assay. Strain TA98 was utilized as the tester strain [32]. The following modifications to this method were employed: 500 µl of urine were used for extraction and the loaded XAD-2 columns were washed with 100 µl methylene chloride. The evaporated eluate was dissolved in 0.75 µl DMSO. The test was performed with 10, 25, 50,

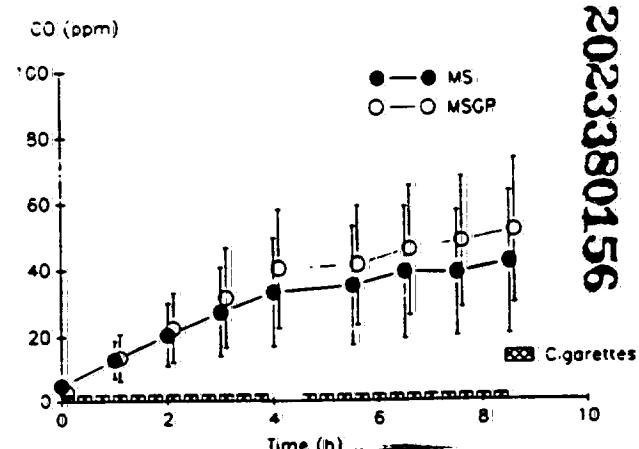
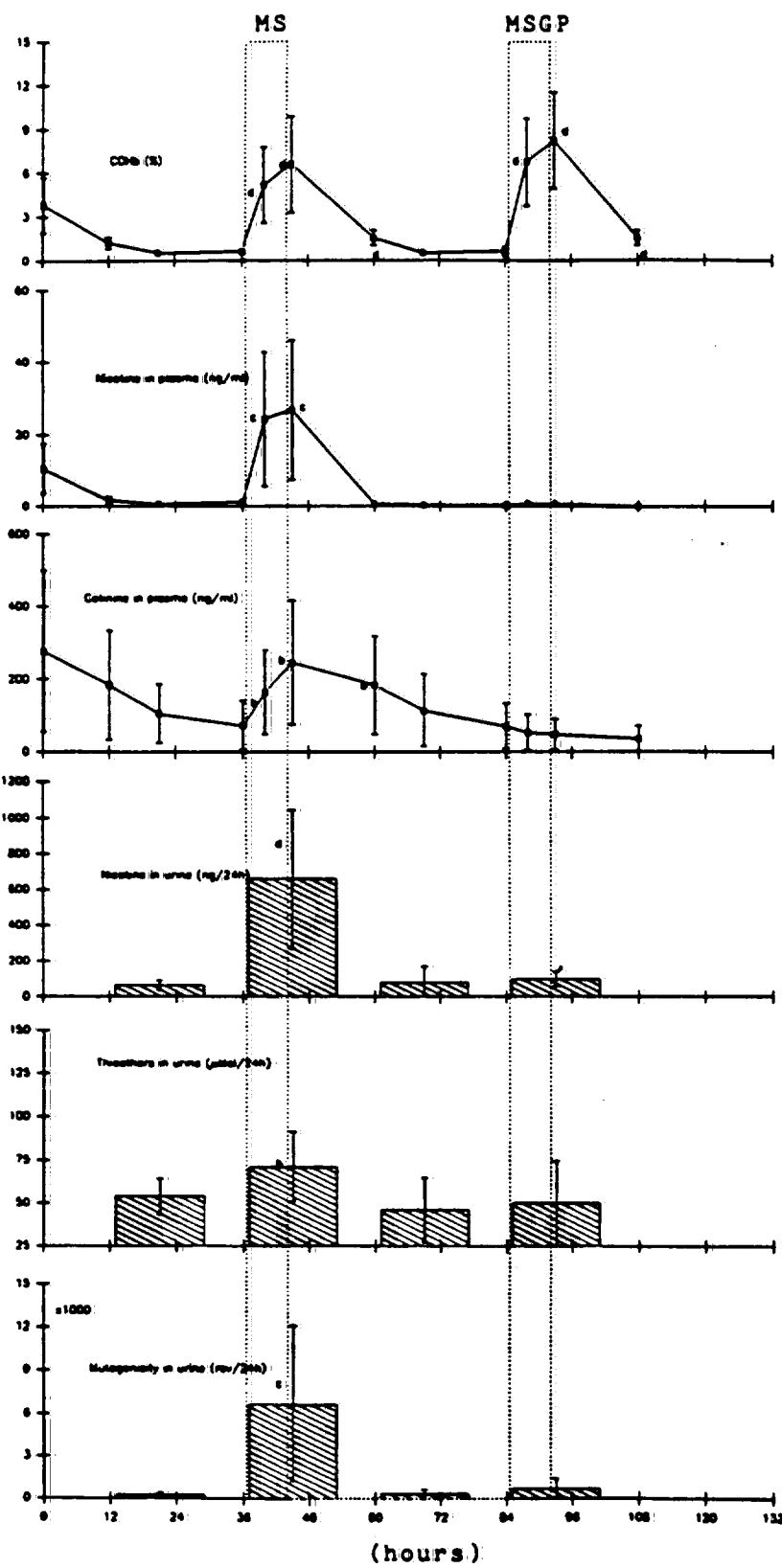


Fig. 1. Carbon monoxide in expired air after smoking whole (MS) and gas phase mainstream smoke (MSGP) during Study 1. (Means with standard deviation bars)

2023380157



**Fig. 2.** Time-courses of the biochemical parameters measured in smokers during Study 1. (Means with standard deviation bars). MS = Whole mainstream smoke; MSGP = Gaseous phase of mainstream smoke; statistical significance:  
 a:  $P < 0.10$ ; b:  $P < 0.05$ ; c:  $P < 0.01$ ; d:  $P < 0.001$ .

and 75 µl aliquots of this extract. These aliquots were equivalent to 6.7, 16.7, 33.3, and 50 ml of urine, respectively. The spontaneous mutation rates in the presence of S-9 mix were 35 to 55 revertants/plate.

**Statistical analysis.** Student's *t*-test for paired samples (exposed vs. non-exposed) was applied. For blood and plasma parameters, the level in the respective morning sample before start of the exposure was used as non-exposed reference value. In case of urine parameters, the level in the 24-h urine of the previous control day was used as a non-exposed reference value.

## Results

### Study 1 (active smoking)

In order to compare the uptake of tobacco smoke constituents from MS and filtered MS (gaseous phase), it was necessary to ensure that at least as much gas phase was inhaled by the smoker under the filter conditions as during active smoking. To be on the safe side, subjects were encouraged to slightly "oversmoke" the gas phase MS smoke. This is seen in the results presented in Fig. 1. On the average, smokers attained higher CO levels in their exhaled breath with gas phase smoking conditions. Although this parameter showed a high inter-individual variation, each subject attained or exceeded the CO levels observed under normal smoking conditions. This increase was consistent with that observed in COHb levels in these subjects (Fig. 2). In contrast, however, no elevation was noted in plasma nicotine levels during gas phase inhalation and plasma cotinine levels continued to decline after gas phase smoking. These results were paralleled by measurements of urinary nicotine under these smoking conditions. This strongly indicates that nicotine was not taken up under gas phase smoking conditions. Both urinary thioether and urinary mutagenicity

were slightly but no significantly increased after gas phase smoking. Urinary mutagenic activity after gas phase smoking was, on the average, less than 1/10 of that observed after smoking whole mainstream smoke and not significantly increased in comparison to that of the control days (Days 1 and 3). Positive Ames tests with clear dose-response relationships were only observed with urine extracts from Day 2 when the subjects smoked the whole MS. Means, standard deviations and ranges of the maximal mutation rates (revertants/plate) were as follows: Day 1 (no smoking): 63 ± 11 (45–97), Day 2 (whole MS smoking): 226 ± 125 (85–589), Day 3 (no smoking): 61 ± 13 (37–84), Day 4 (gas phase MS smoking): 67 ± 13 (47–92).

### Study 2 (passive smoking)

The time-weighted average concentrations of ETS constituents in the exposure room from Day 2 to Day 5 are summarized in Table 1. The substances measured included respirable particulate matter (RSP) and five species of polycyclic aromatic hydrocarbons (PAH). A smoking-related increase is clearly seen in all parameters measured. Some particulate matter constituents [RSP, benzo(a)pyrene (BaP), benzo(e)pyrene (BeP), benzo(a)-anthracene (BaA)] show higher concentrations on Day 5 than on Day 3, although the same number of cigarettes (120) was smoked according to the same time schedule. The reason for this is not quite clear. The ventilation conditions in the room were rather poor, with high temperatures (28–35°C) and a high relative humidity (60–90%). This could have affected the concentrations measured in the room air. The conditions were, however, comparable throughout the study.

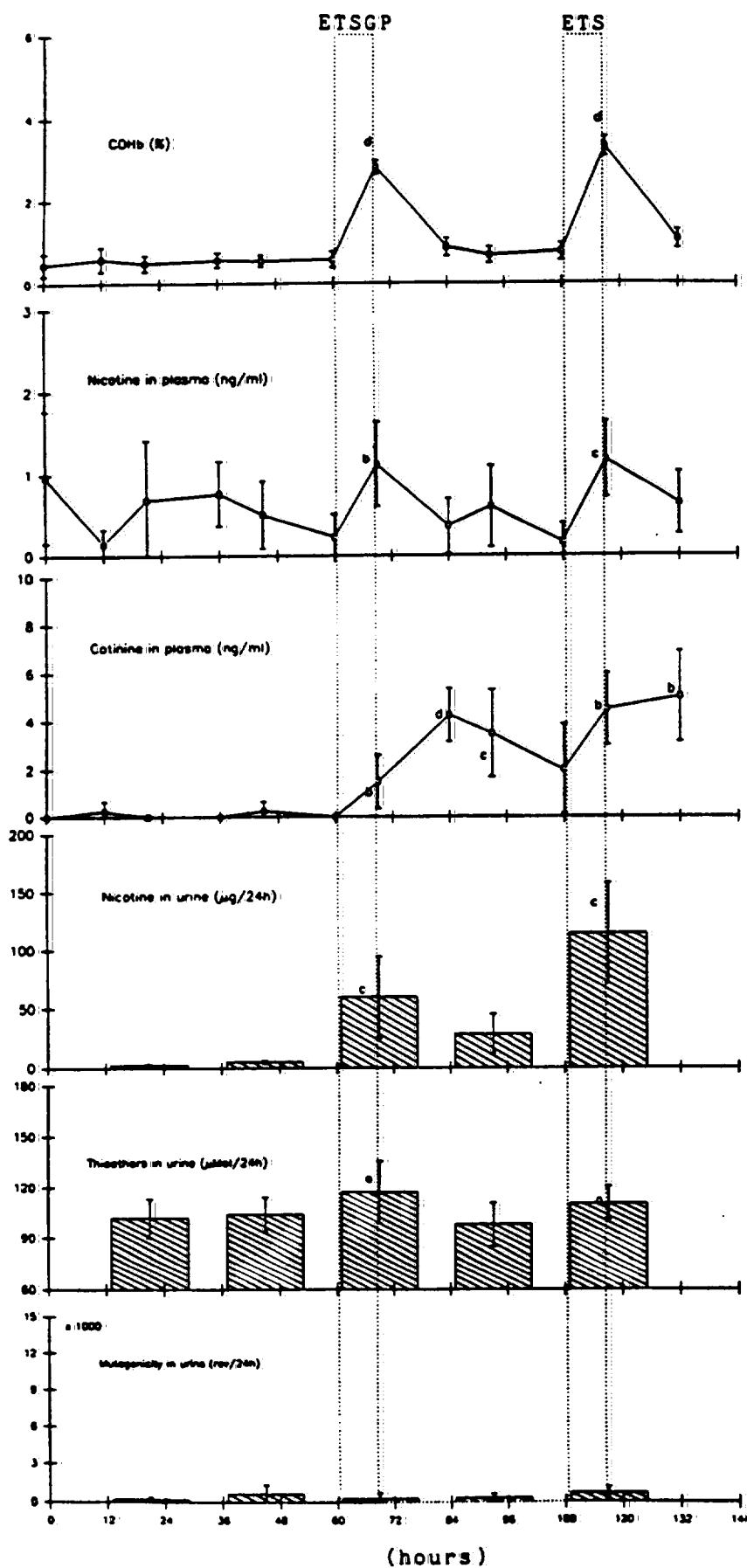
Phenanthrene and pyrene were included in both the particulate matter and gaseous phase section of Table 1, although they are more abundant in the gas phase of ETS. The background levels for both of them are rather high, particularly for the volatile part. Tobacco smoke does not substantially increase the concentrations of these two compounds in the room air.

The biomonitoring results for the non-smokers in Study 2 are shown in Fig. 3. For both types of exposure (gas phase ETS and whole ETS), increases are seen for COHb, nicotine and cotinine in plasma and for urinary nicotine and thioether excretion. The increases in COHb, nicotine and cotinine are statistically significant when compared to the pre-exposure levels. Increase in urinary thioether excretion is only of borderline significance ( $P < 0.10$ ). No significant changes were observed for urinary mutagenicity. No dose-response relationship was found in the Ames test with any of the urine extracts. Means, standard deviations and ranges of the maximal mutation rates (revertants/plate) were as follows: Day 1 (no exposure): 54 ± 10 (35–71), Day 2 (sham-exposure): 58 ± 10 (42–77), Day 3 (gas phase ETS exposure): 57 ± 12 (40–77), Day 4 (sham-exposure): 54 ± 9 (41–71), Day 5 (whole ETS exposure): 62 ± 11 (48–91). After exposure to whole ETS, urinary mutagenicity seems to be slightly elevated. However, a similar amount of excreted mutagenic activity was measured on Day 2, when the subjects

Table 1. Air monitoring in the experimental room. Data are time-weighted averages for the 8-h exposure sessions

Day	2	3	4	5
Cigarettes/8 h	—	120	—	120
<b>Particulate Matter</b>				
RSP (µg/m³)	77	3181	78	4091
BaP (ng/m³)	0.2	21.5	0.3	26.7
BeP (ng/m³)	0.8	21.5	0.8	24.9
BaA (ng/m³)	2.1	18.9	1.1	26.2
Phenanthrene (ng/m³)	3.7	6.8	1.8	7.4
Pyrene (ng/m³)	0.6	17.6	0.7	20.5
<b>Gaseous Phase</b>				
CO (ppm)	1.4	24	2.0	24
NOx (ppb)	38	422	56	449
Formaldehyde (µg/m³)	3	48	3	49
Nicotine (µg/m³)	4	71	6	71
Phenanthrene (ng/m³)	138	154	nd*	258
Pyrene (ng/m³)	29	24	nd	25

\* Not determined



**Fig. 3. Time-courses of the biochemical parameters measured in passive smokers during Study 2. (Means with standard deviation bars).**  
**ETS = Whole environmental tobacco smoke;**  
**ETSGP = Gaseous phase of environmental tobacco smoke;**  
**statistical significance: a:  $P < 0.10$ : b:  $P < 0.05$ : c:  $P < 0.01$ : d:  $P < 0.001$**

2023380159

were not exposed to ETS (609 rev/24 h on Day 2 vs 648 rev/24 h on Day 5). The increases in plasma nicotine and urinary thioether excretion after gas phase ETS and whole ETS show no statistically significant differences. Increase in COHb was slightly but significantly higher after whole ETS exposure (2.6%) than after gas phase ETS exposure (2.2%). Our findings are in line with the fact that the underlying exposure agents, i.e. CO, nicotine and electrophiles (giving rise to thioether formation) are gas phase constituents of ETS. Measurements of cotinine in plasma are unsuitable for such a comparison since carry-over effects occur due to the relatively long half-life of this metabolite [34].

## Discussion

There are many chemical and physical differences between MS and ETS [16] and, as has been discussed by Doll [6], these differences make risk extrapolation from smokers to passive smokers questionable.

Experimental and theoretical considerations also support this view. Chemical analyses of components in ETS [9] and the data presented in this study demonstrate that, in contrast to MS, semi-volatiles are more abundant in the gaseous phase than in the particulate phase of ETS. Other compounds are also distributed to varying degrees between the gas and particle phases of MS and ETS. Some of these and their relative dose ratios are shown in Table 2 [16, 19, 37, 38]. It is clear that the ratio between active and passive smoking depends upon the substances involved and that in the case of passive smoking, gaseous phase compounds are more relevant in the evaluation of uptake than are particle-bound constituents. As seen in Table 2, the uptake of particle phase compounds by active smoking is up to three orders of magnitude higher, whereas uptake of gas phase compounds is only less than one order of magnitude higher than with passive smoking. In addition to the substance related variations in ETS and MS, there are physico-chemical differences which affect uptake. Sidestream smoke is more alkaline than MS, resulting in an increased absorption of nicotine in the oral cavity [17]. Additionally, ETS particles are smaller than MS particles [16, 38]. These factors, together with the different inhalation pattern observed during active and passive smoking, result in much lower particle deposition rates in passive smokers [13, 14]. Results of in vitro tests with ETS and MS suggest that aged ETS is less cytotoxic than fresh MS inhaled by the active smoker [35]. Differences in the biological effects of tobacco smoke exposure could also be expected due to physiological differences between active and passive smokers. An intact clearing mechanism of the respiratory tract, as is usually observed in non-smokers, removes particles more effectively than a damaged one [25]. Smokers have been found to have induced levels in the activities of both toxicifying and detoxifying enzyme systems [27]. Whether in total balance, this has a beneficial effect for the exposed individual, as supposed by Remmer [28], or a harmful effect, is as yet uncertain.

**Table 2. Estimated dose ratio between smoking and passive smoking<sup>a</sup>**

Tobacco smoke constituents	Smoking (S) (20 cig/d) <sup>b</sup>	Passive smoking (PS) (8 h/d) <sup>c</sup>	Dose ratio S/PS
<b>Gaseous phase</b>			
CO (mg)	40–400	14.4–96	2.7–4.2
Formaldehyde (mg)	0.4–1.8	0.08–0.4	4–5
Volatile nitrosamines (µg)	0.05–1.0	0.03–0.4	1.5–2.5
Benzene (µg)	200–1200	40–400	3–5
<b>Particulate matter</b>			
Particles (mg)	75–300	0.024–0.24	1250–3000
Nicotine (mg) <sup>d</sup>	7.5–30	0.08–0.4	75–90
Benzo(a)pyrene (µg)	0.15–0.75	0.001–0.011	70–150
Cadmium (µg)	1.5	0.001–0.014	110–1500
Tobacco specific nitrosamines (µg)	4.5–45	0.002–0.010	2300–4500

<sup>a</sup> Data are compiled from References 16, 19, 37, 38

<sup>b</sup> Assumed deposition rate for particulate matter: 75% (14)

<sup>c</sup> Assumed breathing volume: 0.5 m<sup>3</sup>/h; assumed deposition rate for particulate matter: 11% (13)

<sup>d</sup> Nicotine is particle-bound in MS and a gas phase constituent in ETS (7)

The present results demonstrate that the gaseous phase and the particulate phase of both ETS and MS quite differently affect the biological intake measures assessed in this study. As expected, COHb was found to increase after both gaseous phase and whole smoke exposure in both active and passive smokers. In our investigation, COHb was used to validate the uptake of gas phase and whole smoke under the different exposure conditions. Non-smokers showed a significantly higher increase in COHb after whole ETS exposure (2.6%) when compared to gas phase ETS exposure (2.2%). Since the CO concentration during the 8-h exposure period was similar in both cases (24 ppm), it can be assumed that the non-smokers breathed less deeply when wearing face masks on Day 3. On the other hand, smokers inhaled more intensively when smoking the gas phase mainstream smoke (6.5% vs 8.2% COHb; see also Fig. 1 for CO in expired air). In our view, these relatively small differences in inhalation do not substantially affect the other parameters.

In non-smokers, marginal but similar increases in plasma nicotine concentrations were found after gas phase (1.1 ng/ml) and whole ETS exposure (1.2 ng/ml). Increased urinary nicotine excretion is found after both gas phase and whole ETS exposure, with a higher increase following whole ETS exposure. Three factors could have contributed to the higher increase: (a) nicotine is not completely excreted when the second exposure starts, (b) the subjects inhale more deeply on Day 5 (see COHb values, Fig. 3), (c) part of the nicotine in ETS is also particle bound. However, the data are consistent with the fact that nicotine is mainly a vapour phase constituent in ETS [7]. This is also in accordance with the timecourse of the cotinine plasma levels as ob-

2023380160

served for the non-smokers in Study 2 (Fig. 3). In contrast, nicotine in plasma and urine and cotinine in plasma of smokers were found to be elevated only when smoking whole MS but not gas phase MS. This is in full agreement with the fact that nicotine in MS is almost exclusively bound to smoke particles. The percentage of nicotine in the vapour phase of MS (1–5%) increases with the pH of the smoke and should be higher in cigarettes made from dark tobacco [4]. In our sample only one subject smoked cigarettes of this type. Interestingly, we found the highest increase in plasma nicotine in this individual (2 ng/ml). This increase, however, was very low compared to that after whole MS smoking. Therefore, nicotine and cotinine in smokers reflect smoke particle exposure, whereas in passive smokers these parameters indicate exposure mainly to tobacco smoke vapour phase.

In previous investigations we found an increase in urinary thioether excretion after extremely high ETS exposure under carefully controlled dietary conditions [33]. It is assumed that electrophilic compounds taken up from tobacco smoke give rise to thioether formation [39]. The chemical nature of the smoking- and passive smoking-related thioethers in urine and the electrophiles in tobacco smoke responsible for their formation are unknown. In this investigation we found a similar increase in urinary thioether excretion after exposure to gaseous phase and whole ETS. This confirms our earlier results [33] and indicates that the underlying electrophiles must be gas phase constituents of ETS. In contrast, a significant increase in thioether excretion was only found in smokers when they smoked whole MS. After smoking gas phase MS, only a slight but statistically not significant elevation in thioether excretion was observed. When the same urine samples were analysed for thioethers in another laboratory the increase in thioether excretion was similar both after whole and gas phase MS. Therefore, it is not possible at present to decide whether the electrophiles in MS are predominantly gas phase or particulate phase constituents. The identification of specific thioethers formed after active and passive smoking could give further information on the tobacco smoke constituents which are responsible for thioether excretion.

Urinary mutagenicity was not significantly increased after exposure to gas phase or whole ETS exposure. Earlier studies reported a slight or no increase in urinary mutagenicity after ETS exposure [3, 15, 18, 24, 32, 36], but only in two of them were the results statistically significant [3, 24]. The mutagenic activity after whole ETS exposure in this study tended to be somewhat elevated. However, a similar increase was observed on Day 2 when no exposure took place. Therefore, in our view the elevation on Day 5 cannot be related to ETS particles but is rather due to methodological and biological variation. Urinary mutagenicity in smokers was significantly increased after smoking whole MS. After gas phase MS smoking, a slightly increased urinary mutagenicity was observed. This leads us to conclude that the elevated urinary mutagenicity in smokers is almost completely caused by inhalation and deposition of MS particles. These results also seem to justify the application of urinary mutagenicity as an indirect measure for tar expo-

sure in smokers [2]. The applicability of this method, however, is limited due to the high inter-individual variation in urinary excretion of mutagens.

Our findings on urinary mutagenicity are consistent with the following considerations: Air samples from tobacco smoke polluted indoor environments have been found to contain mutagenic substances which are bound to ETS particles [18, 22]. In a recent study, Salomaa et al. [31] reported only minor mutagenic activity (less than 10% of the activity found in ETS particulate phase) in the vapour phase of ETS when applying the *S. typhimurium* TA 98/microsome assay to both tobacco smoke fractions. As in smokers, urinary mutagenicity in ETS-exposed individuals could therefore be an indirect measure for ETS particle exposure. Based on the particle concentration during ETS exposure (Table 1), a breathing rate of 0.5 m<sup>3</sup>/h and a deposition rate of 11% [13], it can be estimated that the non-smokers in Study 2 could have taken up about 1.8 mg of ETS particles during the 8-h exposure session on Day 5, compared to about 300 mg of particles taken up by the smokers when smoking the whole MS of 24 cigarettes. The Ames test with urine extracts is by far not sensitive enough to detect this exposure. A micro pre-incubation procedure is reported to be 10 to 20 times more sensitive than the conventional plate incorporation test applied in this study [18]. However, no ETS exposure-related increase in urinary mutagenicity was observed by Kado et al. [18] when using the more sensitive test in a pilot-study under realistic exposure conditions.

The results of the biological monitoring in the present investigation demonstrate that the biomarkers which show ETS exposure-related increases (i.e. COHb, nicotine, cotinine and thioethers), only indicate exposure to the gas phase of ETS. However, in active smokers the biomarkers nicotine and cotinine, as well as urinary mutagenicity, indicate uptake of the particulate matter of tobacco smoke. Urinary mutagenicity, which could be a potential marker also for ETS particle exposure, was found to be unchanged after high ETS exposure applied in this study.

In conclusion, these findings give experimental evidence that for passive smoking, exposure to the gas phase of ETS is more important than to the particulate phase. In contrast to smoking, uptake of tobacco smoke derived particles during passive smoking seems to be very low and not detectable by the present methods available for biomonitoring. Therefore, linear risk extrapolation from smoking to passive smoking based on the common intake variables is not warranted.

**Acknowledgements.** We thank Dr. Glaser and Prof. Grimmer for sampling and analysis of PAH.

#### References

1. Aringer L, Lidums V (1988) The influence of diet and other factors on urinary levels of thioethers. *Int Arch Occup Environ Health* 61: 123–130
2. Benowitz NL, Jacob III P, Kozlowski LT, Yu L (1986) Influence of smoking fewer cigarettes on exposure to tar, nicotine, and carbon monoxide. *New Engl J Med* 315: 1310–1313

2023380161

3. Bos RP, Theuws JLG, Henderson PM (1983) Excretion of mutagens in human urine after passive smoking. *Cancer Lett* 19: 85-90
4. Brunnemann KD, Hoffmann D (1974) The pH of tobacco smoke. *Fd Cosmet Toxicol* 12: 115-124
5. Conner JM (1986) Development of a method for estimating the contribution of environmental tobacco smoke (ETS) to indoor respirable suspended particles. Paper presented at the 40th Tobacco Chemists' Research Conference
6. Doll R (1986) Lung cancer: observed and expected changes in incidence from active and passive smoking. Abstracts of Lectures, Symposia and Free Communications of the 14th vol 1. Int Cancer Congress, Budapest
7. Eudy LW, Thorne FA, Heaner DL, Green CR, Ingebretsen BJ (1985) Studies on the vapor-particulate phase distribution of the environmental nicotine. Paper presented at the 39th Tobacco Chemists' Research Conference, Montreal, Canada, November
8. Grimmer G, Naujack K-W, Detlbarn G (1987) Gaschromatographic determination of polycyclic aromatic hydrocarbons, aza-arenes, aromatic amines in sidestream smoke of cigarettes. *Toxicol Lett* 35: 117-124
9. Guerin MR, Higgins CE, Griest WM (1987) The analysis of particulate and vapour phases of tobacco smoke. In: Environmental carcinogens. In: O'Neill IK, Brunnemann KD, Dodet B, Hoffmann D (eds) Methods of analysis and exposure measurements. vol 9 - Passive smoking. IARC Sci Publ No 81, pp 115-139
10. Heinemann G, Schievelbein H, Richter F (1984) Die analytische und diagnostische Validität der Bestimmung von Carboxyhämoglobin im Blut und Kohlenmonoxid in der Atemluft von Rauchern und Nichtrauchern. *J Clin Chem Clin Biochem* 22: 229-235
11. Heinonen T, Kytoniemi V, Sorsa M, Vainio H (1983) Urinary excretion of thioethers among low-tar and medium-tar cigarette smokers. *Int Arch Occup Environ Health* 52: 11-16
12. Hengen N, Hengen M (1978) Gas-liquid chromatographic determination of nicotine and cotinine in plasma. *Clin Chem* 24: 50-53
13. Hiller FC, McCusker KT, Mazumder MK, Wilson JD, Bone RC (1982) Deposition of sidestream cigarette smoke in human respiratory tract. *Am Rev Respir Dis* 125: 406-408
14. Hinds W, First MW, Huber GL, Shea JW (1983) A method for measuring respiratory deposition of cigarette smoke during smoking. *Am Ind Hyg Assoc J* 44: 113-118
15. Husgafvel-Pursiainen K, Sorsa M, Engström K, Einistö P (1987) Passive smoking at work: biochemical and biological measures of exposure to environmental tobacco smoke. *Int Arch Occup Environ Health* 59: 337-345
16. IARC (International Agency for Research on Cancer) (1986) IARC Monographs on the evaluation of the carcinogenic risk of chemicals to humans. vol. 38. Tobacco Smoking. IARC, Lyon
17. Jarczyk L, Scherer G, v. Maltzan C, Luu HT, Adlikofler F (1989) Comparison of intake of nicotine from environmental tobacco smoke between nose and mouth breathers. *Environ Int* 15: 35-40
18. Kado NY, Tesluk SJ, Mammond SK, Woskie SR, Samuels SJ, Schenker MB (1987) Use of a *Salmonella* micro pre-incubation procedure for studying personal exposure to mutagens in environmental tobacco smoke: pilot study of urine and airborne mutagenicity from passive smoking. In: Sandin SS, DeMarini DM, Mass MJ, Moore MM, Mumford JL (eds) Short-term bioassays in the analysis of complex environmental mixtures V. *Environ Sci Res* 36: 375-390
19. Klus H, Begutter H (1987) Environmental tobacco smoke in real life situations. Poster presented at the 4th Int Conf on Indoor Air Quality and Climate, Berlin, 17-21 August 1987
20. Kuwata K, Uebori M, Yamasaki H, Kuge Y (1983) Determination of aliphatic aldehydes in air by liquid chromatography. *Anal Chem* 55: 2013-2016
21. Langone J, Gjika HB, Van Yunakis H (1973) Nicotine and its metabolites: radioimmunoassay for nicotine and cotinine. *Biochem* 12: 5025-5030
22. Ling PI, Löfroth G, Lewthas J (1987) Mutagenic determination of passive smoking. *Toxicol Lett* 35: 147-151
23. Martin F, Hoepfner I, Scherer G, Adlikofler F, Detlbarn G, Grimmer G (1989) Urinary excretion of hydroxy-phenanthrenes after intake of polycyclic aromatic hydrocarbons. *Environ Int* 15: 41-47
24. Mohtashamipur E, Müller G, Norpoth K, Endrikat M, Stücker W (1987) Urinary excretion of mutagens in passive smokers. *Toxicol Lett* 35: 141-146
25. Nakhosteen JA, Lindemann L, Vicira J (1982) Mucociliary clearance. *Disch Med Wochenschr* 107: 1713-1716
26. Odgen MW (1986) Improved gas chromatographic quantitation of trace levels of environmental nicotine. Paper presented at the 40th Tobacco Chemists' Research Conference
27. Pelkonen O, Nebert DW (1982) Metabolism of polycyclic aromatic hydrocarbons: etiologic role in carcinogenesis. *Pharmacol Rev* 34: 189-222
28. Remmert H (1987) Passively inhaled tobacco smoke: a challenge to toxicology and preventive medicine. *Arch Toxicol* 61: 89-104
29. Repace JL, Lowrey AH (1985) A quantitative estimate of non-smokers' lung cancer risk from passive smoking. *Environ Int* 11: 3-22
30. Russell MAH (1987) Estimation of smoke dosage and mortality of non-smokers from environmental tobacco smoke. *Toxicol Lett* 35: 9-18
31. Salomaa S, Tuominen J, Skyttä E (1988) Genotoxicity and PAC analysis of particulate and vapour phases of environmental tobacco smoke. *Mutat Res* 204: 173-183
32. Scherer G, Westphal K, Biber A, Hoepfner I, Adlikofler F (1987) Urinary mutagenicity after controlled exposure to environmental tobacco smoke (ETS). *Toxicol Lett* 35: 135-140
33. Scherer G, Westphal K, Adlikofler F, Sorsa M (1989) Biomonitoring of exposure to potentially mutagenic substances from environmental tobacco smoke. *Environ Int* 15: 49-56
34. Sepkovic DW, Haley NJ, Hoffmann D (1986) Elimination from the body of tobacco products by smokers and passive smokers (letter). *JAMA* 265: 863
35. Sonnenfeld G, Wilson DM (1987) The effect of smoke age and dilution on the cytotoxicity of sidestream (passive) smoke. *Toxicol Lett* 35: 89-94
36. Sorsa M, Einistö P, Husgafvel-Pursiainen K, Järventaus H, Kivistö H, Peltonen Y, Tuomi T, Valkonen S (1985) Passive and active exposure to cigarette smoke in a smoking experiment. *J Toxicol Environ Health* 16: 523-534
37. Sterling TD, Dimich H (1982) Indoor byproduct levels of tobacco smoke. A critical review of the literature. *J Air Pollut Control Assoc* 32: 250-259
38. US Department of Health and Human Services (1986) The health consequences of involuntary smoking. A report of the surgeon general US Government Printing Office, Washington DC
39. Van Doorn R, Bos RP, Leijdekkers CM, Wagenaars-Zegers MAP, Theuws JLG, Henderson PT (1979) Thioether concentration and mutagenicity of urine from cigarette smokers. *Int Arch Occup Environ Health* 43: 159-166

2023380162

**Proceedings of the ASHRAE Conference**

# **IAQ '86**

**Managing Indoor Air for Health  
and Energy Conservation**

**April 20-23, 1986  
Atlanta, Georgia, U.S.A.**



**American Society of Heating, Refrigerating  
and Air-Conditioning Engineers, Inc.**

2023380163

# ASSESSING THE IMPACT OF ENVIRONMENTAL TOBACCO SMOKE ON INDOOR AIR QUALITY: CURRENT STATUS

C.W. Nystrom, Ph.D.    C.R. Green, Ph.D.

## ABSTRACT

The impact of environmental tobacco smoke (ETS) on indoor air quality depends primarily on an assessment of exposure of nonsmokers to smoke constituents. ETS is a dynamic aerosol which exhibits both spatial and temporal variations, losing volatile components from its particles and changing in particulate size over time. The ratios of sidestream to mainstream tobacco smoke concentrations are inappropriately used for assessment of the impact of ETS because these ratios are affected by cigarette moisture content, filter ventilation, porosity of the paper, puff volume by the smoker, etc. Concentrations anticipated in indoor air for CO, NH<sub>3</sub>, HCN, nicotine, and phenol derived solely from ETS are well below threshold limit values established for the workplace. Also, exposure to N-nitrosodimethylamine and benzo[a]pyrene in ETS is low when compared to other environmental sources. There is no satisfactory particulate phase marker for ETS and finding a suitable marker or combination of markers is a formidable challenge for future research.

## INTRODUCTION

Since environmental tobacco smoke (ETS) can produce annoyance and irritation to both the smoker as well as the nonsmoker, its impact on indoor air quality has long been recognized. Under most conditions, the normal ventilation required to maintain a comfortable indoor environment for other reasons is sufficient to prevent annoyance or irritation for most people. The provision of additional ventilation where a large number of smokers are present in a small area has been standard operating practice to maintain comfortable indoor air quality.

Concern among nonsmokers about the possible effects of inhaling other people's cigarette smoke emerged following a 1971 speech by then Surgeon General Jesse Steinfeld, who called for a ban on smoking in public places. Steinfeld said: "...evidence is accumulating that the nonsmoker may have untoward effects from the pollution his smoking neighbor forces upon him" (Steinfeld 1971). In 1974, a workshop organized by Dr. Ragnar Rylander of the Universities of Geneva and Gothenburg was attended by scientists from around the world to consider the health consequences of ETS. These scientists acknowledged the annoying and irritating effects of ETS but did not conclude that ETS is a hazard to nonsmokers (Rylander 1974).

Early attempts to estimate the exposure of nonsmokers to ETS based upon nicotine measurements suggested that a nonsmoker would inhale the equivalent of 1/1000th to 1/100th of the mainstream smoke from a filter cigarette per hour (Hinds and First 1975). At this exposure rate, it would take four days for a nonsmoker to inhale the equivalent of the smoke from a single cigarette.

Although this interpretation of the data may still be valid today, we now know that the estimation, based on nicotine in the atmosphere, represents only the nicotine exposure per se. This will be discussed in more detail later.

Charles W. Nystrom, Senior Staff R&D Scientist; and Charles R. Green, Principal Scientist are in the Research and Development Department, R.J. Reynolds Tobacco Company, Winston-Salem, North Carolina, 27102.

2023380164

Some researchers have determined that compounds suspected of significant biological activity are present in larger quantities in sidestream than in mainstream smoke. To some scientists this finding did not necessarily reflect a serious hazard because of the substantial dilution expected as these compounds diffused into the indoor environment. However, other scientists speculate that the higher levels in sidestream smoke does pose a serious health hazard to the nonsmoker.

Evidence has been obtained that demonstrates that nicotine is taken up by nonsmokers exposed to ETS: Small quantities of cotinine (a major metabolite of nicotine) was found in urine, blood, and saliva of nonsmokers. A variety of studies in chambers and indoor environments have been carried out. Estimates made by several investigators suggest that cotinine levels found in body fluids indicate smoke uptake equivalent to a few cigarettes per day by nonsmokers. However, at a recent workshop on ETS it was estimated that 1/10th cigarette per day was more realistic (Rylander 1983). Also, during the past ten years, a number of epidemiological studies on the alleged health effects of ETS have been carried out and their merits debated in the literature. Although it is beyond the scope of this paper to discuss these studies; they are discussed in reports of two recent international symposia (Rylander 1983; Wynder 1984). Participants in both symposia pointed out that the lack of adequate actual exposure measurements to ETS by the nonsmoking population under normal everyday conditions precludes any meaningful evaluations of the effects of ETS in these epidemiological studies.

During this same period that ETS came under discussion, indoor air quality also became a topic for detailed scientific study. Traditionally, the scientific community had directed its efforts to control outdoor air pollution. However, specific examples of indoor air contamination at concentrations sufficient to produce health effects were detected. Contaminants of concern include radon, carbon monoxide, nitrogen dioxide, formaldehyde, asbestos fibers, microorganisms, and aeroallergens. Potential problems were compounded by the need to conserve energy as energy costs increased. As a result, homes were insulated better and natural air exchange with outside air was reduced. Scientists pointed out that a large portion of the population spends 90% of their time indoors. Studies have shown that outdoor concentrations of pollutants may be poor indicators of the individual's indoor exposure. However, efforts to assess problems associated with indoor air pollution are limited by insufficient information about the number of people exposed, the pattern and severity of exposure, and the health consequences of exposure. In evaluating the impact of ETS on indoor air quality, it is necessary to identify and quantify tobacco smoke components released into indoor air. Also, the dynamic relationship of ETS to other constituents in indoor air, is ultimately required.

#### DESCRIPTION OF ETS

Environmental tobacco smoke is a mixture of both sidestream smoke (SS) and exhaled mainstream smoke (EMS). In the literature "sidestream smoke" is often used synonymously with ETS, however no evidence exists to indicate these are equivalent. This paper presents information showing that SS is not an adequate or reliable surrogate for ETS and such use of SS is inappropriate. The composition of ETS is complex, unstable, and is dependent on the chemical and physical properties of the mixture of SS and EMS over time. It is also dependent on the proportions of both and on the environment into which they are released (volume of space, ventilation rate, absorbency of surfaces, etc.). From the moment of formation, ETS components may change substantially with time (the "aging" process).

The relative contributions of SS and EMS to ETS have not been definitively established. However, a rough estimate can be made from the data obtained by First (1984) with unfiltered cigarettes. In these studies, MS and EMS particulate phases were collected on separate filters, extracted with methanol, and quantitatively determined by the UV absorbance (400 nm). Based on the difference between the MS and the EMS filters, a mean pulmonary deposition of approximately 50% for MS was calculated. Although partial chemical composition of MS, but not of EMS was reported; it was not possible to determine how the MS particulate phase changed as a result of inhalation. While data for SS compositions were presented, the methods of SS collection and analysis were not described. Assuming First's data are valid (and assuming a 50% retention of MS), the contribution of EMS and freshly generated (unaged) SS to ETS can be roughly estimated; e.g., SS would contribute approximately 87% of the particles of unaged ETS, 80% of the "tar," 78% of the nicotine, 88% of the benzo[a]pyrene, and 90% of the CO. For some compounds, the contribution to ETS may be even greater than calculated here; e.g., it has been demonstrated that about 90% of MS nicotine is retained during inhalation. Consequently, more than 90% of the nicotine found in ETS may originate during the smolder of a cigarette.

Based on First's estimates, we have chosen to place our initial emphasis on the properties of SS and follow what happens to it as it diffuses into the indoor atmosphere.

2023380165

## COMPARISON OF MAINSTREAM (MS) AND SIDESTREAM (SS) SMOKE FORMATION

The conditions during the formation of MS and SS smoke has been investigated in great detail by means of a variety of techniques including incorporation of radioactive tracers into smoke, determination of gas and coal temperatures of the cigarette during the smoking cycle, and the determinations of concentrations of individual gases such as CO, CO<sub>2</sub>, H<sub>2</sub>, O<sub>2</sub>, and propane, around the burning coal, (Baker 1975, 1980, 1981a, 1981b, 1982, 1984; Baker and Crellin 1977; Johnson et al. 1973; Jenkins et al. 1975, 1977a, 1977b). The burning cigarette is shown diagrammatically in Figure 1. The smoke formation process is dynamic and conditions change during the puff and smolder periods. To illustrate differences, temperature of both solid and gases within regions of the cigarette coal as reported by Baker (1975) are shown at 1 s into the puff in Figure 2 and again 3 s after the completion of the puff in Figure 3. In general, the temperatures are somewhat lower during the smolder period and will decrease even further as the smolder period (up to 58 s) continues. In Figure 4, the CO concentration in the burning zone of the cigarette is shown at different times during the puff/smolder cycle (Baker 1975). In Figure 5, the CO distribution is shown during the smolder period both inside the cigarette and the area above the cigarette (Baker 1977). In Figure 6, the temperature above the smoldering cigarette is shown (Neurath 1966). Note that the gas plume has a significant temperature (200°C) at 13 mm above the cigarette. Again, the reader is referred to the previously cited papers for more detail on this subject.

Mainstream smoke particles are thought to arise primarily from materials formed in the peripheral portion of the pyrolysis/distillation zone behind the burning coal (see Figure 1). As these materials, primarily gases, are drawn towards the smoker's mouth by the puff, a portion condenses to form the particulate phase of the MS smoke aerosol. The constituents of the SS smoke plume are believed to be formed in both the axial and peripheral region of the pyrolysis/distillation zone. These gaseous constituents diffuse out of the cigarette primarily at a point about 0-4 mm behind the paper burn line where a portion of this vapor condenses to form the SS particles. The vapor leaving the pyrolysis/distillation region of the cigarette to the SS plume is subjected to greater dilution and more rapid temperature losses than that pulled through the tobacco rod to form MS (Baker 1982). These conditions would be expected to favor the formation of smaller SS particles, which indeed has been shown to be the case as discussed later. The major portion of the gaseous sidestream components originates from the cigarette coal, rising in a fairly well-defined column centered about 3 mm in front of the paper burn line. This gas plume is primarily composed of CO, CO<sub>2</sub>, H<sub>2</sub>, and H<sub>2</sub>O. In addition to the SS plume, small quantities of gases such as CO and NH<sub>3</sub> diffuse through the cigarette paper into the environment during both the puff and smolder periods.

### PARTICLE SIZE OF ETS

The fate of ETS particulate matter in the indoor environment depends on, among other things, its particle size distribution initially. The size distribution of SS particles was determined over a period of time in an 0.5-m<sup>3</sup> chamber. Size distribution and concentration of SS particles were determined with a combination of an optical particle counter, an electrostatic mobility analyzer system, and a condensation nucleus counter (Ingebretsen and Sears 1985). Their results were as follows: At low mass loadings (several  $\mu\text{g}/\text{m}^3$ ) typically observed in ETS, the particles have a mean diameter of 0.098 micron, and a mass median diameter of 0.185 micron. While higher mass loadings >220  $\mu\text{g}/\text{m}^3$ , the ETS particles have a median diameter of 0.141, and a mass median diameter of 0.21 micron. In all cases there is an insignificant mass >1.0 micron size and a large number of particles <0.10 micron. Figure 7 shows an increase in the average mass diameter over time as determined by optical particle counter measurements. Based on these and other supporting data, Ingebretsen and Sears (1985) conclude that the smaller average mass diameter in the early portion of the curve is a result of evaporation of volatile constituents. Subsequently, the average mass diameter increases very slowly due to a combination of coagulation and deposition of smaller particles on surfaces. Surface deposition of ETS is the main route of removal in a static situation and is a function of particle size, mixing rate, and room size and shape. It is estimated that 20-30% of the particulate matter in "fresh" ETS is lost by evaporation of volatile constituents.

### PROBLEMS IN COLLECTING REPRESENTATIVE SS SMOKE SAMPLES

MS smoke is readily collected by a variety of techniques which can be reproduced from one laboratory to another. This is possible because techniques have been devised to essentially duplicate the process of puffing on the cigarette. The burning end of the cigarette is held under conditions comparable to those occurring when a cigarette is smoked by a smoker. Although small changes in MS smoke chemistry may occur as a result of the collection procedure,

2023380166

It is generally accepted that the smoke collected at the butt end of the cigarette is representative of the MS smoke received by the smoker. However, in the case of SS smoke, the analytical problem is much more complex because the collection process interferes with the normal burning process of the cigarette and with its diffusion into the atmosphere. These interferences could include the reduction in the diffusion of the SS gases into the atmosphere, temperature and associated chemical changes decreases as the SS plume diffuses into the atmosphere, and/or interferences in the normal burning process in the cigarette coal. Several of these problems have been reported with the apparatus used to collect sidestream smoke (Brown et al. 1980; Harris and Hayes 1978; Neurath et al. 1966; Sukama 1983). Perhaps, the apparatus most frequently employed in investigating SS composition is the modified Neurath chamber shown in Figure B. The main problem is that the temperature inside this collection device increases considerably. Although adjustments can be made in the airflow to reproduce the MS FTC "tar" delivery rate, it is possible that the combustion/pyrolysis products may undergo quite different changes during mixing and increasing temperatures than normally occurs in SS smoke formation. Recently Brunnemann et al. (1980) compared SS smoke in a hermetically sealed chamber, and found only 67-87% of the expected amount of N-nitrosodimethylamine (NDMA) as in the modified Neurath chamber. Stehlik et al. (1981) found 25% less NDMA when SS smoke was allowed to diffuse into a sealed chamber compared with that in the modified Neurath chamber. Hence, smoke chemistry based on these collection techniques may not reflect the chemical composition of ETS to which the nonsmoker is exposed.

#### FACTORS THAT AFFECT SS/MS RATIO

The use of the SS/MS ratio for smoke constituents has been a common practice which evolved in an attempt to draw attention to the ETS issue. Although this practice may have provided useful information in the early days, it has little practical relevance since the ratio may be modified by a number of factors. For example, the levels of some cigarette smoke components are greater in the SS smoke because more tobacco is burned during smolder than during the puff periods. Some additional factors are discussed in the following text.

#### Moisture Content

The moisture content does affect the amount of tobacco burned during the puff and smolder periods. In Table 1, the quantity of tobacco burned during the puff and smolder periods for a low moisture (4.2%) and a normal or control (12.6%) tobacco are used to calculate the ratio of the puff/smolder period. A much greater portion of the tobacco rod is consumed during the smolder period under normal conditions versus those in the test. When the tobacco moisture is low, the quantity of tobacco burned during the smolder period is significantly increased; ratio of smolder/puff was 2.52 compared to 1.74 for control. Of course, the tobacco industry makes a great effort to keep its products at the optimum moisture level but such an effect could be produced if the cigarette packs are left opened, or stored for a long time in hot dry climates, etc.

#### Air Dilution

Air dilution from filter perforations affects the SS/MS ratio causing major reductions in all MS components. This source of air dilution is characteristic of many commercial cigarettes. Browne et al. (1980) investigated the effect of this source of air dilution on some major MS and SS smoke components. Their results show that the SS/MS ratio for "tar", CO, and nicotine increases as air dilution rate increases, see Table 2.

#### Paper Porosity

Another factor that affects the SS/MS ratio is the porosity of the cigarette paper. The results of a study on the effect of paper porosity conducted by Harris and Taylor (1980) are shown in Table 3. It can be seen that the ratio of tobacco burned during the smolder to that burned during the puff period increases as the paper porosity increases. This would be expected to increase the SS/MS ratio of most constituents similar to air dilution.

#### Puff Volume

Another factor that influences the SS/MS ratio is the puff volume used by the smoker. This was also investigated by Browne et al. (1980). In Table 4 are data from Browne et al. (1980) and Perfetti et al. (1983). As the puff volume increases, the SS/MS ratio for "tar", CO, and nicotine decrease.

2023380162

## SS/MS Ratios in Commercial Cigarettes

Other factors could be cited, but the point is that the SS/MS ratio is not constant and can vary considerably. To illustrate this, the results of analyses of some typical commercial cigarettes smoked under standard Federal Trade Commission test conditions for selected MS and SS constituents are shown in Tables 5 and 6. These results, obtained by Harris and Hayes (1978), are not necessarily representative of all commercial cigarettes, but they do give an overview of what one might expect if a systematic survey were made. Note that, although wide variations in SS/MS ratios are obtained, the actual SS values do not vary appreciably.

### Effect of Dilution as SS Diffuses into Indoor Air

The anticipated impact of dilution in room air on the concentrations of specific SS smoke constituents as they diffused into the indoor environment is illustrated in the following: Table 7 shows average values calculated from the SS smoke values shown in Tables 5 and 6. In addition, average values of four commercial cigarettes as reported by Adams et al. (1985) for benzo[a]pyrene (B[a]P), catechol, and N-nitrosodimethylamine (NDMA), are included. To illustrate the effect of dilution, it is assumed that indicated smoke constituents undergo a dilution similar to that of the particulate matter from ETS. Obviously this assumption is not a totally accurate assessment of what will happen, but it does give an estimation of what quantities of these constituents might be expected in indoor air if they behave similarly to particulate matter. The values of 25 and 250  $\mu\text{g}$  were selected since these quantities seem to represent realistic conditions. In the only study that has attempted to correct for other sources of respirable suspended particles (RSP), results by Spengler et al. (1985) show that an increase of about 20-25  $\mu\text{g}/\text{m}^3$  in RSP is observed in a home with one smoker versus homes with no smokers. As an additional reference point, the current threshold limit values (TLVs) recommended by the American Conference of Governmental Industrial Hygienists for exposure in the workplace for CO, nicotine, NH<sub>3</sub>, HCN and phenol, TLVs values are shown in Table 7. The concentration of each of these constituents found in these studies are extremely low and well below the TLVs for exposure in the workplace.

It is also of interest to compare the daily exposure of the nonsmoker to B[a]P and NDMA at an assumed exposure of ETS particles (25  $\mu\text{g}/\text{m}^3$ ) with those from other sources. These have been calculated and are shown in Table 8 and compared to values reported in the literature for exposure from air pollution (range shown is for several different cities) and dietary intake (Sawicki et al. 1960; Dennis et al. 1983; Preussmann 1983).

## DECAY OF SPECIFIC ETS CONSTITUENTS IN AN ENVIRONMENTAL CHAMBER

Investigations in an 18- $\text{m}^3$  stainless steel test chamber have been described by Heavner et al. (1985). This chamber has the capability to be operated in six modes. Techniques for the measurement of temperature, humidity, and air exchange rates are provided. The chamber may be operated manually or under control of a computer, which can also log data directly. Analyzers directly coupled to the chamber include continuous monitors for measurement of CO, CO<sub>2</sub>, NO, NO<sub>2</sub>, and FID response. Particle-measuring instrumentation for size distribution, number concentration, and mass concentration is also provided. An atmospheric pressure chemical ionization MS/MS system (APCI MS/MS system) with the capability to measure up to eight selected compounds (Szadkowski et al. 1976) has been used to detect only compounds in the gas phase. Generally, the analytical systems are calibrated with National Bureau of Standards (NBS) traceable gases or certified permeation tubes. The chamber is provided with remote ignition and extinguishing of the cigarette. In the experimental studies reported, the cigarette is smoked according to standard FTC conditions, a 2-s 35-ml puff once per minute. The MS smoke is exhausted from the chamber whereas the SS smoke from one cigarette was allowed to diffuse throughout the chamber. The chamber was operated in the static mode; therefore, the concentration obtained was the maximum possible from one cigarette.

In Figure 9, the changes in nicotine and particulate matter over time are shown when the environmental chamber is operated in the static mode (no air exchange). Nicotine was determined by the APCI MS/MS system and particulate matter by the piezoelectric balance. Note that nicotine and particulate matter decay at different rates.

In Figure 10, the changes in nicotine and pyridine under static chamber conditions are shown. Neither the nicotine nor pyridine follow the same rate of decay.

Other studies are investigating the effects of factors such as mixing rate and air exchange on the decay of ETS constituents. The static conditions used to obtain the results reported are not representative of the real-world situations, and the differences in decay observed between

2023380168

nicotine, pyridine, and particles may not be as great when other factors are considered. Nevertheless, these results suggest that extreme caution must be used in attempts to extrapolate from observed concentration of one constituent to another.

#### NICOTINE AS A MARKER FOR ETS OR ETS EXPOSURE

Nicotine is a unique component of tobacco smoke, and tobacco products are generally the only source of this alkaloid in indoor air. Nicotine is a major component of tobacco smoke and has been considered by several authors as the largest component of ETS particulate matter other than water. Recent studies by Eudy et al. (1985) have established that nicotine is not a component of ETS particulate phase but is in the gas phase of ETS.

Nicotine determinations in indoor air therefore simply reflect the exposure to the concentration of nicotine per se. Muramatsu et al. (1984) recently conducted a survey of nicotine concentrations in a number of real-life conditions; their results are shown in Table 9.

#### USE OF COTININE IN BODY FLUIDS AS A MARKER FOR ETS EXPOSURE

Other investigators have used the cotinine concentration in body fluids to estimate nicotine uptake and to calculate exposure in cigarette equivalents. Although the relationship between nicotine uptake and cotinine concentrations in body fluids has been established for the smoker, a similar relationship between nicotine uptake at dose levels comparable to that received by the nonsmoker from ETS has not been established. Two studies have been reported in which this was attempted. However, in both cases, the ETS concentrations are much higher than normally encountered by the nonsmoker in real-life situations. More importantly, the variability in the concentration of cotinine in the body fluids indicates it will be very difficult to estimate the nicotine uptake.

In Table 10, the concentrations of specific ETS constituents in a test chamber employed by Hoffmann et al. (1984) in the exposure of individuals to ETS are shown. The average values for cotinine in body fluids obtained at different exposure levels are shown in Table 11. Unfortunately, only the average values are given, and no indication of the differences between individuals for the cotinine concentrations in body fluids is presented.

Johnson et al. (1985) exposed ten healthy subjects to ETS in a climatic chamber for a period of three hours. Since the CO averaged 25-30 ppm and the nicotine was about 280  $\mu\text{g}/\text{m}^3$ , it is apparent that high ETS concentrations were used. The results of cotinine analysis in body fluids are summarized in Table 12. Two different methods of analysis for cotinine were employed. Note the large variability in the concentrations of cotinine in urine observed in individuals presumably exposed to the same quantity of nicotine. These results suggest that the individual variability in the biological handling of nicotine will greatly influence the quantity of cotinine in the urine. Hence, it is very difficult to relate ETS nicotine exposure to cotinine in body fluids. Once again, it should be noted that even if this can be done, it will only be an indicator of nicotine uptake and may or may not reflect the exposure to other ETS constituents. More work is needed in this area.

#### USE OF CARBON MONOXIDE AS MARKER FOR ETS

Carbon monoxide is a major SS gas-phase constituent and, hence, is a major component contributed by ETS to indoor air. Historically, it has been the most widely used chemical marker for monitoring ETS exposure. This no doubt results from the fact that readily available techniques for measurement of both the concentration of CO in air and the uptake or absorption of CO by humans are available. The latter is possible since the concentration of carboxyhemoglobin ( $\text{COHb}$ ) can be readily measured.

Despite the ready availability of analytical techniques, it has been difficult to discern the effect of CO from ETS except under extreme conditions of experimentally designed exposure. This difficulty is due to interference from other sources of CO. If you recall, it was estimated earlier that as one allows for atmospheric dilution, the CO from ETS would be highly diluted. It was estimated that conditions that would result in a particulate concentration of 250  $\mu\text{g}/\text{m}^3$  would result in about 979  $\mu\text{g}/\text{m}^3$  or roughly 1 mg/m<sup>3</sup> of CO. This is equivalent to slightly less than 1 ppm. Thus, although it is a major component of ETS, the CO from ETS would generally be expected to contribute less than 1 ppm CO above background levels from other sources. Since a variety of other sources, including heating, cooking, and vehicle exhaust, often create background concentrations in the ppm range, it is usually difficult to show a specific increase in CO due to ETS except in controlled chamber studies (Dahms et al. 1981; Pinn et al. 1978; Russell et al. 1973; Seppanen et al. 1977; Seppanen 1977) where high levels of ETS are used.

6910520233816

The uptake of CO by nonsmokers is low and the COHb concentration in the blood (COHb) in the blood. Although small increases in COHb can be demonstrated in individuals exposed to high levels of ETS in controlled chamber exposure studies, it is not possible to show consistent increases in the COHb of nonsmokers exposed to ETS due to the contributions from other sources. Also, the concentrations of COHb in nonsmokers in general are relatively low (Carsky et al. 1980; Cole 1975; Einbrodt et al. 1976; Janzon et al. 1981; Stewart et al. 1974; Stewart et al. 1976; Wald et al. 1981). Hence, it can be concluded that the CO is an ineffective marker for ETS exposure and also that ETS CO has an insignificant effect on the COHb concentration in nonsmokers.

### MARKER FOR ETS PARTICLES

CO and nicotine are representative components of the gas phase of ETS. However, there is a definite need for a marker to separate the contribution of ETS particles from other particulate sources. Since nicotine is not a viable marker for ETS particles, this will be a formidable task. Work is underway in several research laboratories attempting to identify a marker for ETS. Such a marker or markers is essential for determination of the exposure of nonsmokers.

### SIGNIFICANCE OF THE LARGE NUMBER OF CONSTITUENTS IN CIGARETTE SMOKE

As the controversy over ETS has evolved, several individuals have expressed concern about the large number of constituents identified in tobacco smoke. Although SS has not been investigated as extensively as MS, it is anticipated that the same compounds are present in both. Most of these SS compounds are present at concentrations well below levels required to produce a biological effect. In fact, it is evident that we are all exposed to similar numbers of compounds from other environmental sources. If the same detailed and aggressive techniques were applied to our food supply, a similar number of compounds could possibly be identified; e.g., in a recent review on coffee, cocoa, and tea flavors by Flament (1983), it was reported that approximately 620 compounds have been identified in coffee flavor, 380 compounds in tea flavor, and 420 compounds in cocoa flavor. It was also noted that nearly 4000 volatile compounds were listed as components of food products.

### CONCLUSIONS

ETS is recognized to contain a number of constituents capable of impacting indoor air quality. Because of many components with a low odor threshold and its visibility, the presence of ETS is readily detected even at low concentrations. Hence, it may be a marker that reflects poor or inadequate ventilation. ETS is a dynamic material formed primarily from the sidestream of the smoldering cigarette and its particle size changes due to evaporation of volatile constituents and other processes as it mixes in the indoor atmosphere. Much additional work is needed to establish the true contribution of ETS to specific chemical constituents in the indoor air. The use of SS/MS ratios is inappropriate to assess the impact of ETS because these ratios are affected by moisture content, filter ventilation, porosity of the paper, puff volume by the smoker, etc. It would seem to be more important to determine the concentration of specific constituents of ETS to which the nonsmoker is exposed in the indoor atmosphere. The estimated concentrations for CO, NH<sub>3</sub>, HCN, phenol and nicotine, when anticipated dilution is considered, suggest that they are extremely low when compared to the TLVs established for each of these constituents in the workplace. Estimates of exposure to NDMA and B[a]P at anticipated dilutions in indoor air from ETS is relatively low when compared to exposures from other sources such as dietary intake, air, etc.

At present, there is no satisfactory marker for ETS; especially the particulate phase of ETS. Nicotine is a component of the gas phase of ETS and preliminary evidence indicates that it decays at a different rate than the particulate phase. Finding a satisfactory marker or markers to assess the contribution of ETS to indoor air and for use in exposure assessment are the challenges which must be met in the future before the impact of ETS on indoor air quality can be assessed satisfactorily.

### REFERENCES

- ACGIH. 1984-85. "TLVs: Threshold limit values for chemical substances in the work environment." American Conference of Governmental Industrial Hygienists.
- Adams, J.D.; O'Mara-Adams, K.J.; Hoffmann, D. 1985. "On the mainstream-sidestream distribution of smoke components from commercial cigarettes." 39th Tobacco Chemists' Research Conference. Montreal, PQ.

2023380170

- Aviado, D.M. 1984. "Carbon monoxide as an index of environmental tobacco smoke exposure." Europ. J. Resp. Dis. Suppl., Vol. 65, No. 133, pp. 47-61.
- Baker, R.R. 1975. "Temperature variation within a cigarette combustion coal during the smoking cycle." High Temperature Science, Vol. 7, pp. 236-247.
- Baker, R.R. and Crellin, R.A. 1977. "The diffusion of carbon monoxide out of cigarettes." Beitrage zur Tabakforschung Int., Vol. 9, pp. 131-140.
- Baker, R.R. 1980. "Mechanisms of smoke formation and delivery." Recent Adv. in Tobacco Sci., Vol. 6, pp. 184-224.
- Baker, R.R. 1981a. "Variation of the gas formation regions within a cigarette combustion coal during the smoking cycle." Beitrage zur Tabakforschung Int., Vol. 11, pp. 1-17.
- Baker, R.R. 1981b. "Product formation mechanisms inside a burning cigarette." Prog. Ener. Combust., Vol. 7, pp. 135-153.
- Baker, R.R. 1982. "Variation of sidestream gas formation during the smoking cycle." Beitrage zur Tabakforschung Int., Vol. 11, No. 4, pp. 181-190.
- Baker, R.R. 1984. "The effect of ventilation on cigarette combustion mechanisms." Recent Adv. in Tobacco Sci., Vol. 10, pp. 88-150.
- Browne, C.L.; Keith, C.H.; Allen, R.E. 1980. "The effect of filter ventilation on the yield and composition of mainstream and sidestream smokes." Beitrage zur Tabakforschung Int., Vol. 10, pp. 81-90.
- Brunnemann, K.D. and Hoffmann, D. 1973. "The pH of tobacco smoke." Fd. Cosmet. Toxicol., Vol. 12, pp. 115-124.
- Brunnemann, K.D.; Yu, L.; Hoffmann, D. 1977. "Chemical studies on tobacco smoke. XVII. Assessment of carcinogenic volatile N-nitrosamines in tobacco and in mainstream and sidestream smoke from cigarettes." Cancer Res., Vol. 37, pp. 3218-3222.
- Brunnemann, K.D.; Fink, W.; Moser, F. 1980. "Analysis of volatile N-nitrosamines in mainstream and sidestream smoke from cigarettes by GLC-TEA." Oncology, Vol. 37, pp. 217-222.
- Carsky, J.; Hrnciarova, M.; Rusnakova, S.; Busova, B.; Pajdlhauserova, A. 1980. "Carboxyhemoglobin level in blood donors in Bratislava." Bratisl. Lek. Listy., Vol. 74, pp. 273-278.
- Cole, P.V. 1975. "Comparative effects of atmospheric pollution and cigarette smoking on carboxyhemoglobin levels in man." Nature(London), Vol. 255, pp. 699-701.
- Dahms, T.E.; Bolin, J.F.; Slavin, R.G. 1981. "Passive smoking: Effects on bronchial asthma." Chest, Vol. 80, pp. 530-534.
- Dennis, M.J.; Massey, R.C.; McWeeny, D.J.; Knowles, M.E. 1983. "Analysis of polycyclic aromatic hydrocarbons in UK total diets." Fd. Chem. Toxic., Vol. 21, No. 5, pp. 569-574.
- Einbrodt, M.J.; Rosmanith, J.; Prajsnar, D. 1976. "Der cadmiumgehalt im blut und rauchgewohnheiten." Naturwissenschaften, Vol. 63, p. 148.
- Eudy, L.W.; Thome, F.A.; Heavner, D.L.; Green, C.R.; Ingebretsen, B.J. 1985. "Studies on the vapor-particulate phase distribution of environmental nicotine by selected trapping and detection methods." 39th Tobacco Chemists' Research Conference. Montreal, PQ.
- First, M.W. 1984. "Constituents of sidestream and mainstream tobacco smoke and markers to quantify exposure to them." In Indoor Air & Human Health, R.B. Gammie, S.V. Kaye, and V.A. Jacobs, Eds. Lewis Publishers, Inc. (1985), Proceedings of the 7th Life Sciences Symposium, Knoxville, TN.
- Flament, I. 1983. "Coffee, cocoa and tea flavors: A review of present knowledge." Colloq. Int. Aromes Aliment., pp. 46-81.

- Harke, H.P. 1970. "Zum problem des passiv-rauchens". Muench. Med. Wochenschr., Vol. 112, pp. 2328-2334.
- Harris, J.L. and Hayes, L.E. 1978. "A 20-port sidestream smoke collection system." 32nd Tobacco Chemists' Research Conference. Montreal, PQ.
- Harris, J.L. and Taylor, J.N. 1982. Unpublished data.
- Heavner, D.L.; Thome, F.A.; Eudy, L.W.; Green, C.R. 1985. "A test chamber and instrumentation for the analysis of environmental tobacco smoke (ETS)." 39th Tobacco Chemists' Research Conference. Montreal, PQ.
- Hinds, W.C. and First, M.W. 1975. "Concentrations of nicotine and tobacco smoke in public places." New Engl. J. Med., Vol. 292, p. 844.
- Hoffmann, D.; Haley, M.J.; Adams, J.D.; Brunnemann, K.D. 1984. "Tobacco sidestream smoke: Uptake by nonsmokers." Preventive Med., Vol. 13, pp. 608-617.
- Ingebretsen, B.J. and Sears, S.B. 1985. "Particle size distribution measurements of side stream cigarette smoke." 39th Tobacco Chemists' Research Conference, Montreal, PQ.
- Jenkins Jr., R.W.; Comes, R.A.; Bass, R.T. 1975. "The use of carbon-14 labeled compounds in smoke precursor studies-A review." Recent Adv. in Tobacco Sci., Vol. 1, pp. 1-30.
- Jenkins Jr., R.W.; Frisch, A.F.; MacKinnon, J.G.; Williamson, T.G. 1977. "Cigarette smoke formation studies, Part 4: The dynamic measurement of the axial density of a burning cigarette." Beitrage zur Tabakforschung Int., Vol. 9, pp. 67-71.
- Jenkins Jr., R.W.; Bass, R.T.; Newman, R.H.; Chavis, M.K. 1977. "Cigarette smoke formation studies, Part 5: The effects of the cigarette periphery on mainstream smoke formation." Beitrage zur Tabakforschung Int., Vol. 9, pp. 126-130.
- Janzon, L.; Lindell, S.E.; Trell, E.; Larne, P. 1981. "Smoking habits and carboxyhemoglobin: A cross-sectional study of an urban population of middle-aged men." J. Epidemiol. Commun. Health, Vol. 35, pp. 271-273.
- Johnson, L.C.; Letzel, H.; Kleinschmidt, J. 1985. "Passive smoking under controlled conditions." Int. Arch. Occup. Environ. Health, Vol. 56, pp. 99-110.
- Johnson, W.R.; Hale, R.W.; Nedlock, J.W.; Grubbs, H.J.; Powell, D.H. 1973. "The distribution of products between mainstream and sidestream smoke." Tobacco Sci., Vol. 17, pp. 141-144.
- Klus, H. and Kuhn, H. 1982. "Distribution of various tobacco smoke components in mainstream and sidestream smoke." Beitrage zur Tabakforschung Int., Vol. 11, pp. 229-265.
- Muramatsu, M.; Umemura, S.; Okada, T.; Tomita, H. 1984. "Estimation of personal exposure to tobacco smoke with a newly developed nicotine personal monitor." Environ. Res., Vol. 35, pp. 218-227.
- Neurath, G.; Ehmk, H.; Schneemann, H. 1966. "On the water content of mainstream and side stream smoke." Beitrage zur Tabakforschung Int., Vol. 3, pp. 351-357.
- Perfetti, P.; Townsend, D.; Taylor, J.N. 1983. Unpublished data.
- Pimm, P.E.; Silverman, F.; Shephard, R.J. 1978. "Physiological effects of acute passive exposure to cigarette smoke." Arch. Environ. Health, Vol. 33, pp. 201-213.
- Preussmann, R. 1983. "Occurrence and exposure to N-nitroso compounds and precursors." In N-Nitroso Compounds Occurrence, Biological Effects & Relevance to Human Cancer, J.K. O'Neill, R.C. Von Borstel, C.T. Miller, J. Long, and H. Bartsch, Eds. IARC (1984). Proceedings of the 8th International Symposium on N-nitroso Compounds, Banff, AB. 2023380125
- Russell, M.A.H.; Cole, P.V.; Brown, E. 1973. "Absorption by non-smokers of carbon monoxide from room air polluted by tobacco smoke." Lancet, Vol. 1, pp. 576-579.
- Rylander, R. 1974. "Environmental tobacco smoke effects on the non-smoker: Report from a workshop." Scand. J. Resp. Dis., Vol. 91, pp. 1-90.

Rylander, R.; Peterson, Y.; Snella, M.C. 1984. "ETS-environmental tobacco smoke: Report from a workshop on effects and exposure levels." Geneva, Switzerland. Euro. Jour. Respir. Dis., Vol. 65, pp. 1-147.

Sakuma, H. 1983. "The distribution of cigarette smoke components between mainstream and sidestream smoke. I. Acidic components." Beitrage zur Tabakforschung Int., Vol. 12, pp. 63-71.

Sawicki, E.; Elbert, W.C.; Hauser, T.R.; Fox, F.T.; Stanley, T.W. 1960. "Benzo[a]pyrene content of the air of American communities." Indust. Hygiene Jour., (Dec.), pp. 444-451.

Seppanen, A. and Uusitalo, A.J. 1977. "Carboxyhaemoglobin saturation in relation to smoking and various occupational conditions." Ann. Clin. Res., Vol. 9, pp. 261-268.

Seppanen, A. 1977. "Smoking in closed spaces and its effects on carboxyhaemoglobin saturation of smoking and non-smoking subjects." Ann. Clin. Res., Vol. 9, pp. 281-283.

Spengler, J.D.; Treitman, R.D.; Tosteson, T.D.; Mage, D.T.; Soczek, M.L. 1985. "Personal exposures to respirable particulates and implications for air pollution epidemiology." Environ. Sci. Technol., Vol. 19, pp. 700-707.

Stehlik, G. 1982. "Concentration of dimethylnitrosamine in the air of smoke-filled rooms." Ecotoxicology and Environmental Safety, Vol. 6, pp. 495-500.

Steinfeld, J.L. 1971. Address to the National Interagency Council on Smoking and Health. Washington, DC.

Stewart, R.D.; Baretta, E.D.; Platte, L.R.; Stewart, E.B.; Kalbfleisch, J.H.; Van Yserloo, B.; Rimm, A.A. 1974. "Carboxyhemoglobin levels in American blood donors." JAMA, Vol. 229, pp. 1187-1195.

Stewart, R.D.; Hake, C.L.; Wu, A.; Stewart, T.A.; Kalbfleisch, J.H. 1976. "Carboxyhemoglobin trend in Chicago blood donors, 1970-1974." Arch. Envir. Health, Vol. 31, pp. 280-286.

Szadkowski, D.; Harke, H.P.; Angerer, J. 1976. "Kohlenmonoxidbelastung durch passivrauchen in buroräumen." Inn. Med., Vol. 3, pp. 310-313.

Thome, F.A.; Heavner, D.L.; Ingebrethsen, B.J.; Eudy, L.W.; Green, C.R. 1985. "Calibration and sampling techniques for an atmospheric pressure chemical ionization mass spectrometer coupled to a test chamber for the study of environmental tobacco smoke." 39th Tobacco Chemists' Research Conference. Montreal, PQ.

Wald, M.J.; Idle, M.; Boreham, J.; Bailey, A. 1981. "Carbon monoxide in breath in relation to smoking and carboxyhaemoglobin levels." Thorax, Vol. 36, pp. 366-369.

Wynder, E.L. 1984. "Proceedings of the International Symposium on Medical Perspectives on Passive Smoking. Vienna, Austria. April 9-12. Prev. Med., Vol. 13, pp. 557-756.

TABLE 1

Effect of Moisture Content on The Quantity of Tobacco Burned during Puff and Smolder of Cigarettes

<u>MOISTURE CONTENT (%)</u>	<u>BURN PERIOD</u>	<u>TOBACCO BURNED(mg)</u>	<u>RATIO (SMOLDER/PUFF)</u>
4 (DRY)	SMOLDER	63.0	2.52
	PUFF	25.0	
12.6 (CONTROL)	SMOLDER	48.7	1.74
	PUFF	28.5	

2023380123

TABLE 2

Effect of Filter Ventilation (Air Dilution) on SS/MS Ratio of 'Tar', CO, and Nicotine from Cigarettes Smoked under Standard Conditions (Browne et al. 1980)

VENTILATION No Dilution	SMOKE STREAM SS	'TAR' mg/cig.	RATIO SS/MS	CO mg/cig.	RATIO SS/MS	NICOTINE mg/cig.	RATIO SS/MS
		17.55	0.597			5.94	
33% Dilution	SS	18.97	0.849	49.3	3.82	6.73	4.37
	MS	22.35		12.9		1.54	
48% Dilution	SS	18.96	1.207	58.4	8.85	6.36	5.48
	MS	15.71		6.6		1.16	
83% Dilution	SS	19.19	2.139	56.3	23.5	6.90	12.5
	MS	8.97		2.4		0.55	

TABLE 3

Effect of Cigarette Paper Permeability on Tobacco Burned in Puff and Smolder of Cigarette (Harris and Taylor 1982)

POROSITY (cm/min)	BURN PERIOD SMOLDER	mg BURNED	RATIO
			32.45
7	PUFF	42.38	0.77
	SMOLDER	49.10	
45	PUFF	33.59	1.46

TABLE 4

Effect of Puff Volume on Yield of 'Tar', CO, and Nicotine in Sidestream (SS) and Mainstream (MS) and on SS/MS Ratio

PUFF VOLUME 17.5 ml	SMOKE STREAM SS	'TAR'	RATIO	CO mg/cig.	RATIO SS/MS	NICOTINE mg/cig.	RATIO SS/MS
		mg/cig.	SS/MS				
35 ml	SS	17.55	.597	49.6	2.67	5.94	3.49
	MS	29.38		18.6		1.70	
50 ml	SS	18.64	.544	56.4	2.76	6.12	2.90
	MS	34.27		20.4		2.11	
From Browne et al. (1980)							
20 ml	SS	26.9	1.42			4.27	2.89
	MS	18.9				1.48	
35 ml	SS	25.4	.988			4.33	2.17
	MS	25.7				2.00	
65 ml	SS	22.2	.663			3.73	1.57
	MS	33.5				2.38	
From Perfetti et al. (1983)							

2023380124

TABLE 5

Yields and Sidestream/Mainstream Ratios (SS/MS) of Nicotine, CO, and 'Tar', from Commercial Cigarettes Smoked under Standard Conditions (Harris and Hayes 1978)

BRAND	SMOKE STREAM	NICOTINE	RATIO	FTC 'TAR'	RATIO	CO	RATIO
		mg/cig.	SS/MS	mg/cig.	SS/MS	mg/cig.	SS/MS
A	SS	4.4	3.38	27.0	0.89	64.3	3.33
	MS	1.3		19.2		19.3	
B	SS	4.0	3.64	16.7	0.91	54.2	3.05
	MS	1.1		18.3		17.8	
C	SS	4.4	5.5	15.1	1.40	60.3	3.58
	MS	.8		10.8		16.8	
D	SS	3.9	6.5	15.0	1.69	59.2	5.19
	MS	.6		8.9		11.4	
E	SS	4.2	21.0	10.7	7.13	45.1	
	MS	.2		1.5		2.3	
F	SS	4.2	21.0	10.2	4.86	47.3	
	MS	.2		2.1		3.3	
G	SS	5.6	3.29	19.5	0.90	77.6	
	MS	1.7		21.6		26.2	

TABLE 6

Yields and Sidestream/Mainstream Ratios (SS/MS) of Phenol, HCN, and NH<sub>3</sub> from Commercial Cigarettes Smoked under Standard Conditions (Harris and Hayes 1978)

BRAND	SMOKE STREAM	PHENOL	RATIO	HCN	RATIO	NH <sub>3</sub>	RATIO
		µg/cig.	SS/MS	µg/cig.	SS/MS	mg/cig.	SS/MS
A	SS	61	1.69	140	0.42	6.17	199
	MS	36.1		344		.031	
B	SS	69	2.4	114	0.31	8.40	156
	MS	27.9		363		.054	
C	SS	68	2.65	---	---	5.80	
	MS	25.7		151		.020	290
D	SS	58	3.63	133	0.82	7.86	328
	MS	16.0		163		.024	
E	SS	54	20.0	97	3.34	5.05	1263
	MS	2.7		29		.004	
F	SS	61	9.10	---	---	5.67	
	MS	6.7		17		.005	1134
G	SS	103	1.96	143	0.38	8.30	
	MS	52.5		379		.036	231

2023380125

TABLE 7

Effect of Dilution on Concentration of Individual ETS Components Assuming "Tar" as Reference Material and Comparison to TLVs (ACGIH 1984-85)

<u>SMOKE COMPONENT</u>	<u>TYPICAL SIDESTREAM YIELD</u>	<u>CONCENTRATION</u> <u>ug / m<sup>3</sup></u>		<u>TLV-TWA</u> <u>ug / m<sup>3</sup></u>
'TAR'	14.9 mg	25	250	-----
CO	58.0 mg	97.9	979	55000
NICOTINE	4.39 mg	7.4	74	500
NH <sub>3</sub>	6.75 mg	11.3	113	18000
HCN	71 ug	0.12	1.2	10000(c)
PHENOL	68 ug	0.11	1.1	19000
CATECHOL*	83.5 ug	0.14	1.4	
BAP*	52.5 ng	0.000087**	0.00087	
NDMA*	657 ng	0.00110	0.0110	

\* Based on values reported by Hoffmann et al. (1985).

\*\* This is 87 picograms.

TABLE 8

Comparison of Daily Exposure to NDMA and B[a]P from ETS and from Other Sources

	<u>ETS PARTICULATE (25 ug/m<sup>3</sup>)</u>	<u>FOOD INTAKE</u>	<u>AIR</u>
NDMA	0.0242 ug	1.1 ug (1979) 0.5 ug (1981)	*
B[a]P	0.00174 ug	0.17 ug	0.038-0.41 ug

\* R. Preussmann estimates daily exposure of workers in the rubber industry to be 15-150 ug (NDMA and NMOR).

2023380176

TABLE 9  
NICOTINE IN VARIOUS LIVING ENVIRONMENTS (Muramatsu et al., 1984)

LOCATION	NO. OF SAMPLES	NICOTINE CONCENTRATION µg/m <sup>3</sup>	
		RANGE	AVERAGE
OFFICE A	4	9.34-31.57	19.44
OFFICE B	6	14.60-26.08	22.15
LABORATORY	8	1.76-9.64	5.80
FIVE CONFERENCE ROOMS	5	16.54-53.01	38.73
THREE HOUSES	5	7.61-14.60	11.16
HOSPITAL LOBBY	7	1.89- 5.02	2.98
FOUR HOTEL LOBBIES	5	5.45-18.06	11.18
SEVEN TEA ROOMS	12	15.10-60.89	33.41
FIVE RESTAURANTS	8	7.09-27.81	14.76
THREE STUDENT CAFETERIAS	6	11.59-42.16	26.42
THREE BUS AND RAILWAY WAITING ROOMS	6	10.05-36.43	19.07
FOUR CARS	4	7.73-83.13	47.71
EIGHT TRAINS	8	8.64-26.14	16.42
SEVEN AIRPLANES (DOMESTIC AIRLINE)	7	6.28-28.78	15.18

TABLE 10  
Test Laboratory Conditions for Exposure of Subjects to ETS (Hoffmann et al., 1984)

Size: 16.3 m<sup>3</sup>  
 Temperature: 22 + 1 C  
 Air Exchanges: Six times per hour  
 Pollutants: Sidestream smoke of four concurrently smoked IRI reference cigarettes

<u>ETS Constituent</u>	<u>Concentration</u>
Particulate matter	4,600 µg/m <sup>3</sup>
Nicotine	280 µg/m <sup>3</sup>
Hydrogen cyanide	56 µg/m <sup>3</sup>
Carbon monoxide	25 ppm
NO	0.91 ppm
Formaldehyde	160 µg/m <sup>3</sup>

2023380177

TABLE 11

Cotinine Levels (Summary of Average Values) in Saliva, Serum, and Urine on Volunteers Exposed to ETS (Hoffmann et al. 1984)

<u>Time</u>	<u>Saliva (ng/ml)</u>	<u>Serum (ng/ml)</u>	<u>Urine (ng/mg creatinine)</u>		
	4	4	2	3	4
Baseline	1.0	0.9	14	14	14
I 40	1.1	0.9			
I 60	2.1	1.2	16	21	28
O 30	1.7	1.8			
O 120	2.5	2.9	21	34	46
O 240	2.0	3.3			
O 300	3.5	3.4	21	38	55

Numbers represent room pollution by smoke of 2, 3, or 4 cigarettes.

I = Inside exposure room during pollution.

O = Outside exposure room after leaving the room.

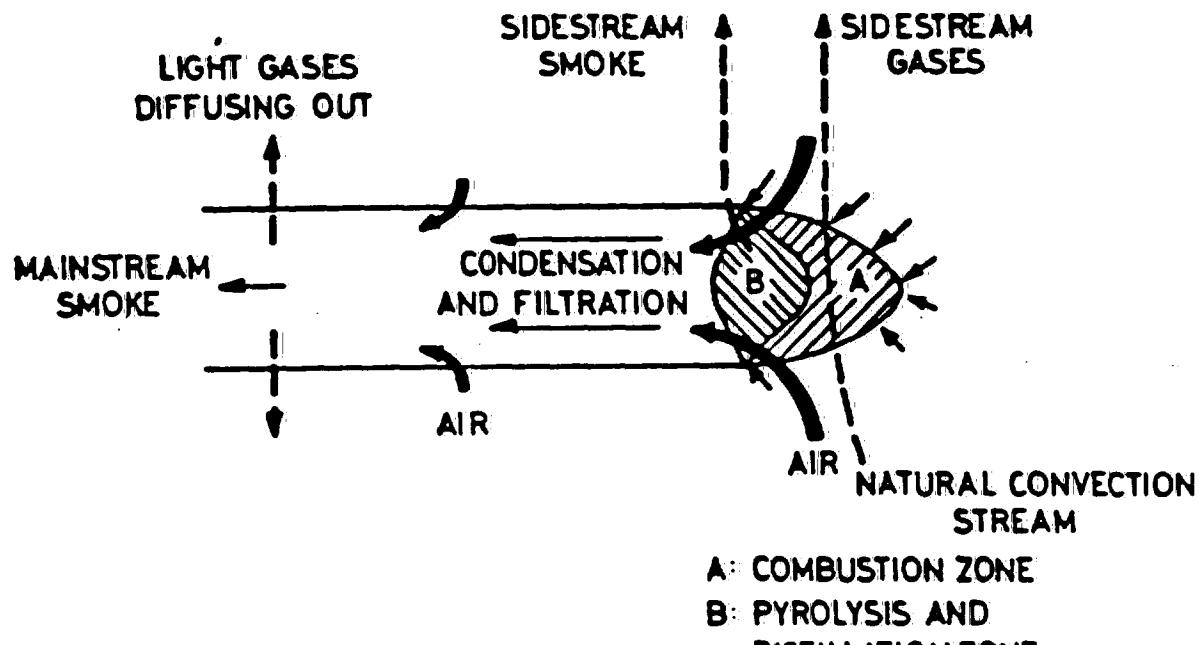
TABLE 12

Cotinine in Body Fluids by Two Methods after Three Hours Exposure to ETS (Johnson et al., 1985)  
(Nicotine 280 µg/m<sup>3</sup>; CO 25-30 ppm)

SOURCE	TIME OF COLLECTION (HOURS)	N	<u>X ± S.D.</u>	
			RADIOIMMUNE ASSAY	GAS CHROMATOGRAPHY
Saliva (ng/ml)	6	8	19.7 ± 4.58	18.72 ± 6.25
Plasma (ng/ml)	4	9	9.00 ± 4.88	8.88 ± 5.31
Urine	24	10	110.21 ± 54.75	83.83 ± 47.3

2023380178

## PUFFING



## SMOLDER

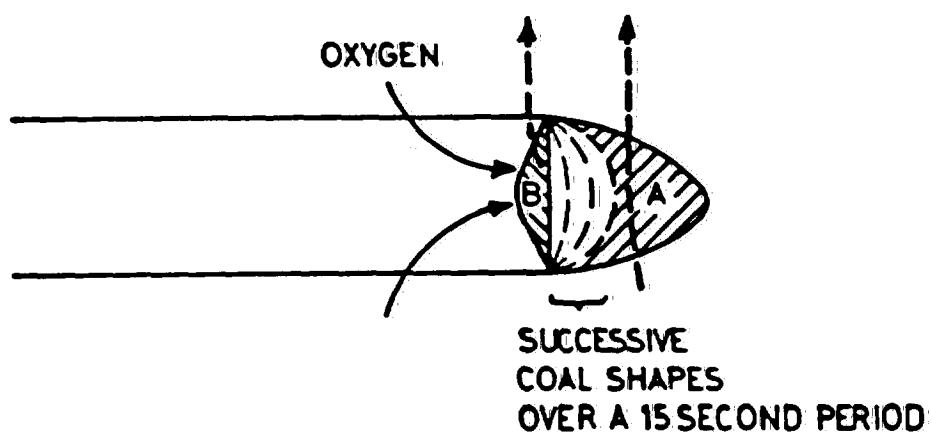


Figure 1. Diagram of the burning cigarette (Baker 1984)

2023380179

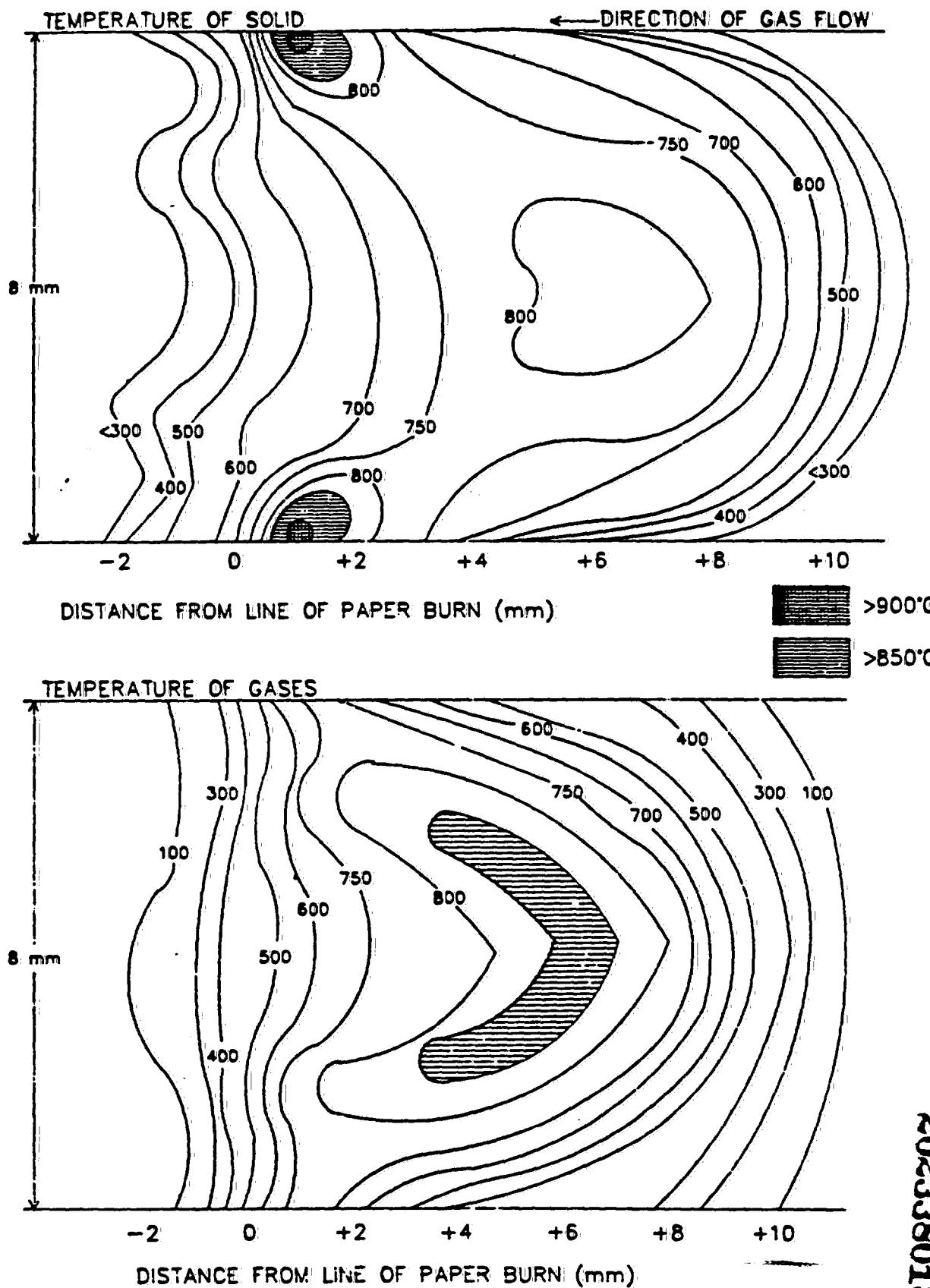


Figure 2. Temperature ( $^{\circ}\text{C}$ ) distribution in the cigarette coal at 1 s after start of a puff (Baker 1975)

2023380180

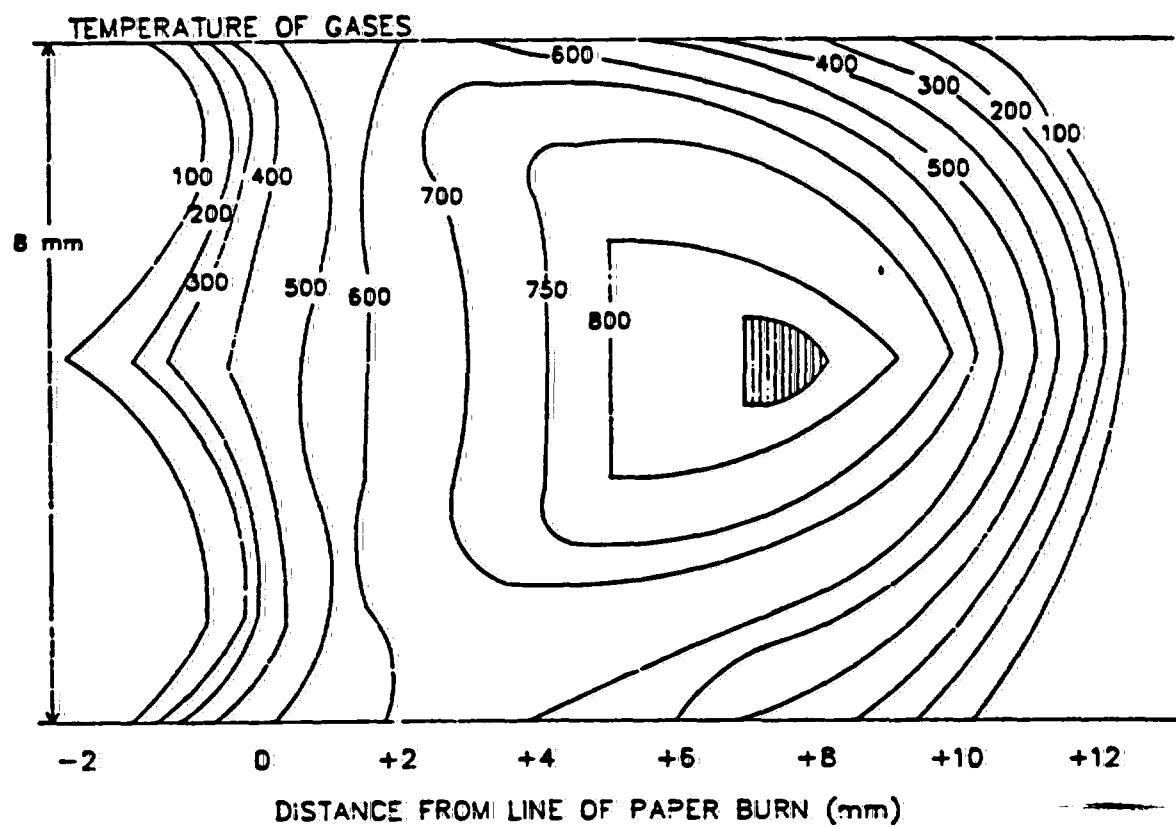
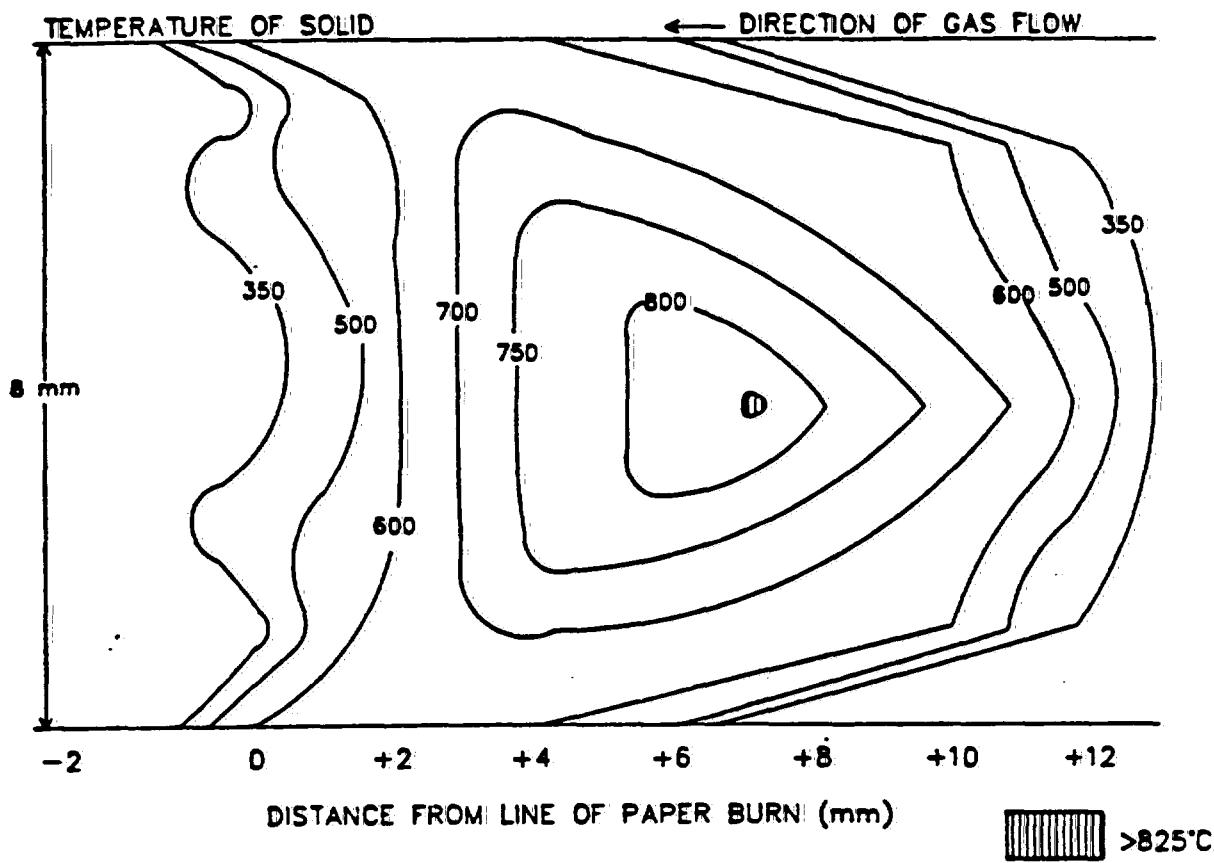
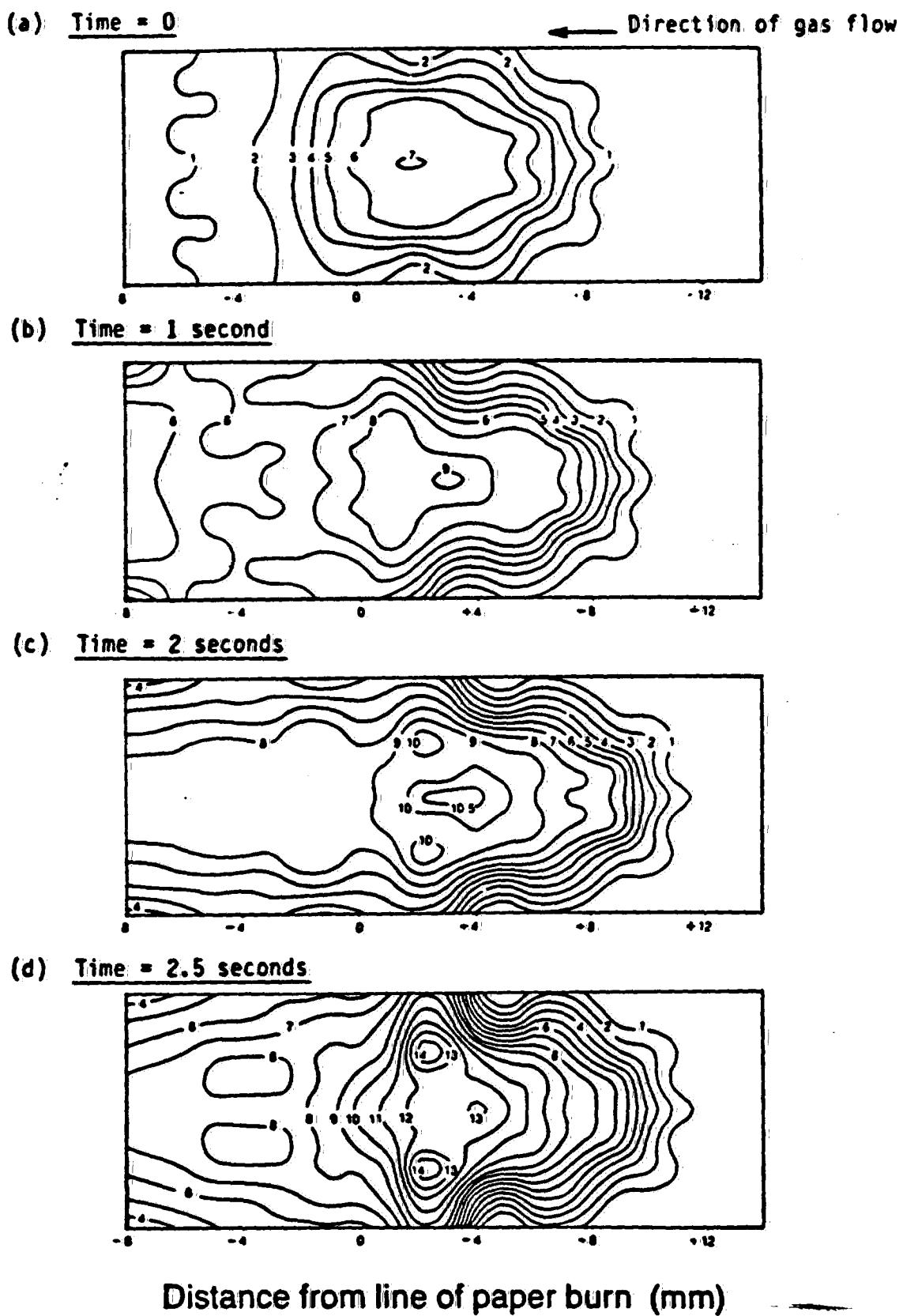


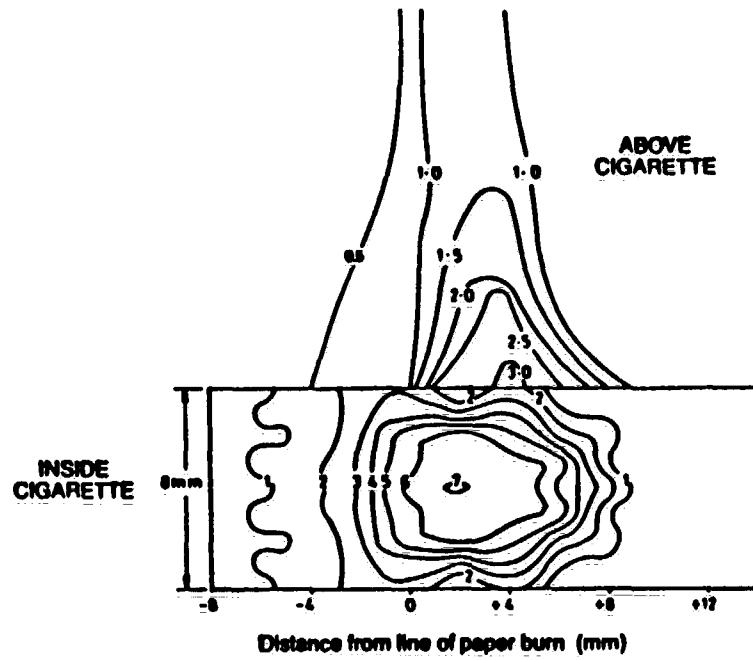
Figure 3. Temperature (°C) distribution in the cigarette coal at 1 s after the end of the puff (Baker 1975).

2023380181



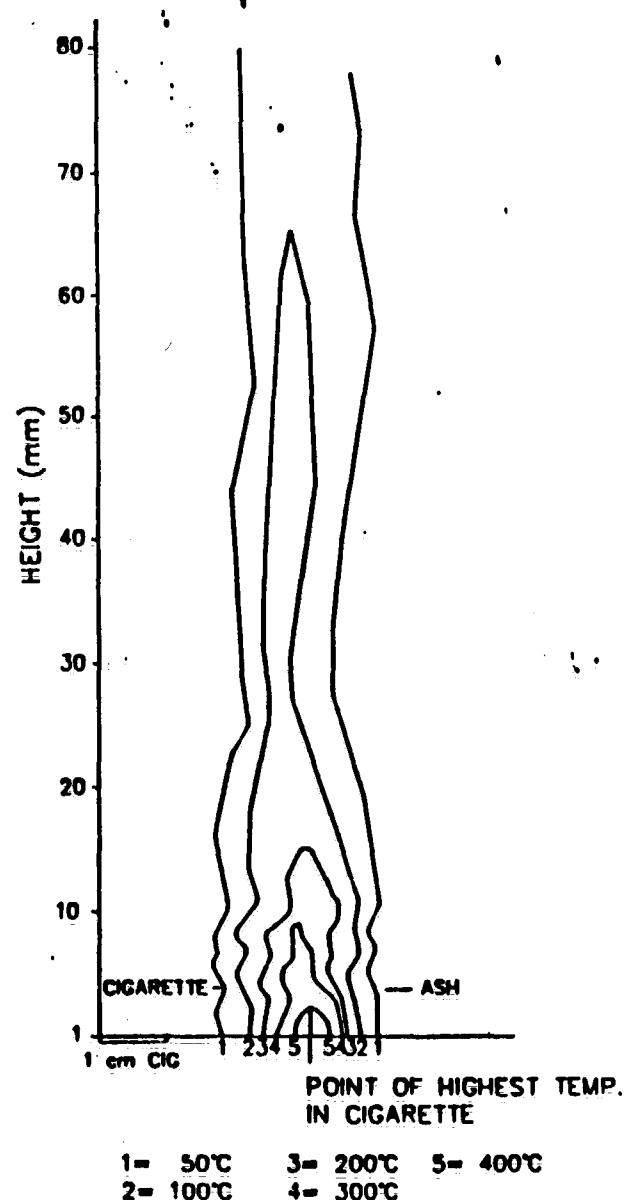
**Figure 4.** Variation in carbon monoxide (RV/V) distribution inside the burning zone after the start of a 2 s puff (Baker 1984)

2023380182

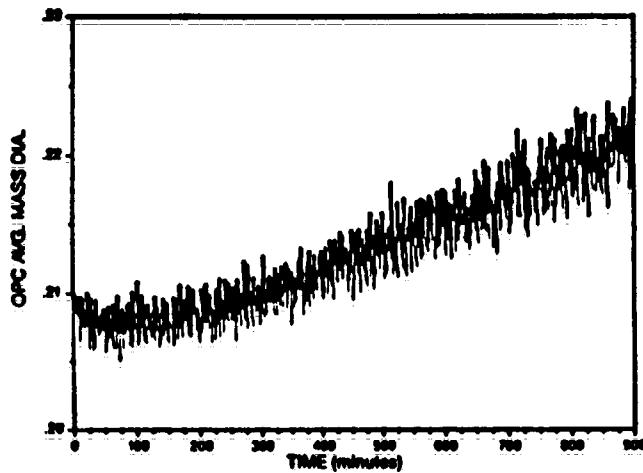


2023380183

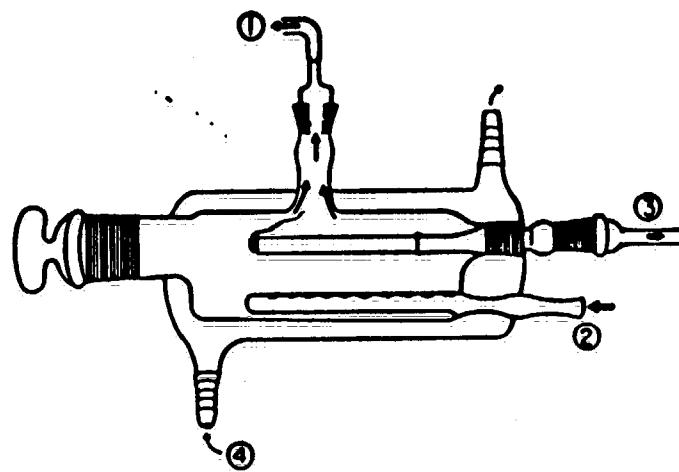
**Figure 5.** Carbon monoxide concentration (ppm) during cigarette smolder (Baker 1984)



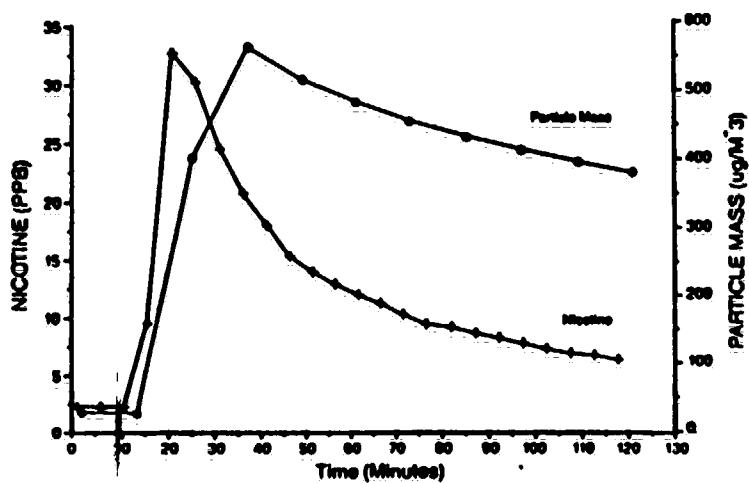
**Figure 6.** Isotherms over a smoldering cigarette (Neurath 1966)



**Figure 7.** Average mass diameter vs. time for particulate phase ( $20 \text{ g/m}^3$ ) of ETS as determined with optical particle counter (Ingebretsen and Sears 1985)

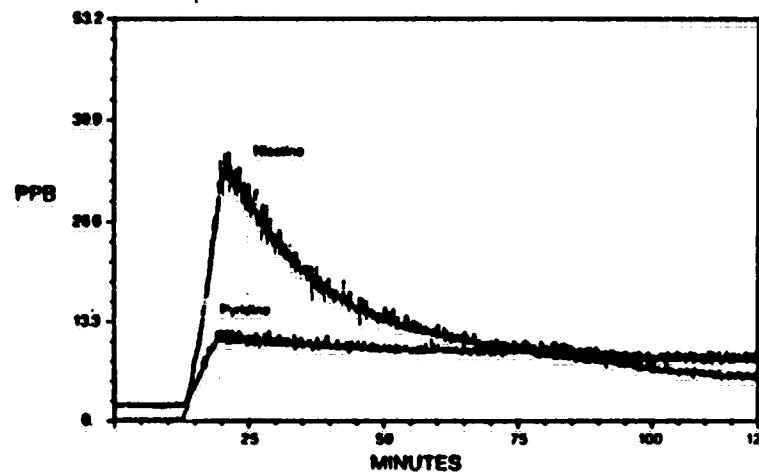


**Figure 8.** Modified Neurath Chamber for collection of sidestream smoke (Brunneman and Hoffman 1974): (1) to trap, (2) air intake, (3) to smoking machine, (4) cooling water intake



**Figure 9.** Comparison of decay of nicotine and particulate matter of ETS in chamber operated in static mode (no air exchange)

2023380184



**Figure 10.** Comparison of decay of nicotine and pyridine of ETS in chamber operated in static mode (no air exchange)

## The Aging of Sidestream Tobacco Smoke Components in Ambient Environments

R. R. Rawbone, W. Burns, and G. Haslett

### Summary

A large number of sidestream smoke components have been measured over a 50-min time period in a well-defined experimental room. The results show a variable rate of decay following smoking which would suggest that extrapolation from a single measured "marker" to other potential smoke components should be performed with caution.

### Introduction

Environmental tobacco smoke is a dynamic aerosol and its characteristics, both physical and chemical, depend on a number of factors; these include the elapsed time since its formation, whether the smoke plume is allowed to fully form before dispersion and the more general dilution within the ambient environment [1]. In terms of a single point sampling site the resultant measurement value will therefore not only depend upon the characteristics of the environment and the number and manner of cigarettes being smoked but also upon both temporal and spatial factors of the sampling position relative to the smoking.

This dynamic nature of the aerosol results not only in a loss of volatile components, including nicotine, from the particles to the vapour phase, but also in a complex and variable behaviour of the individual chemical components which manifests in their exhibiting different decay characteristics. This is of importance in the interpretation of ambient air studies which are generally limited to the measurement of one or two environmental tobacco smoke markers.

The objective of this paper is to demonstrate this variability in decay patterns for a series of chemical measurements over a 50-min period following smoke generation in a well defined experimental room.

### Materials and Methods

Smoke was generated using a modified smoking head from a Battelle rotary smoking machine [2] in a specially constructed room with a volume of about 48,000 litres. The internal walls, ceiling and floor were coated with a sealant paint and there was a single door with no windows, other than a sealed observation port; all other access, including that for electricity supply and air sample collection, was through sealed ducting. During the current studies there was no active ventilation in the room and furniture was kept to a minimum. Temperature and humidity were monitored continuously.

At the start of each experiment 16 cigarettes, with a standard mainstream delivery of 17 mg tar (PMWNF), were smoked on the rotary smoker to the reference conditions of one 35 ml puff of 2 s duration every minute. The mainstream smoke was ducted away and the sidestream smoke, after formation of the plume, mixed into the room by a series of fans. In order to maintain a constant carbon monoxide level in the room throughout a 50-min-study period, as a standard condition, single cigarettes were smoked subsequent to the initial 16 cigarettes being extinguished. The time at which the initial cigarettes were extinguished was also taken as time zero for the commencement of chemical measurement.

Ambient chemistry in the room was measured using the following techniques which have also been employed, for comparative purposes, in a benchtop collection device [3] for the measurement of freshly generated sidestream smoke:

Carbon monoxide was measured continuously using a non-dispersive infra-red analyser (Analytical Development Co., Model RFA/1).

Nicotine, which is distributed between the particulate and vapour phases, was measured as total nicotine by collection into a Tenax trap over 5-min-sampling periods, with subsequent thermal desorption and gas chromatographic analysis (Perkin Elmer, ATD50).

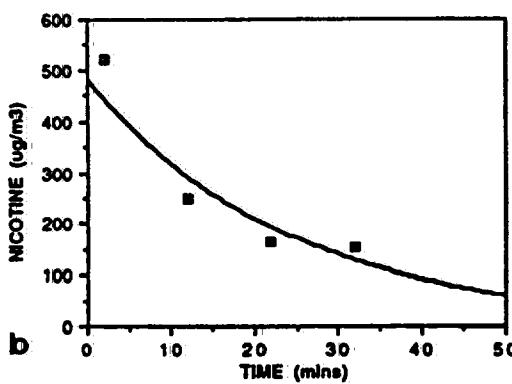
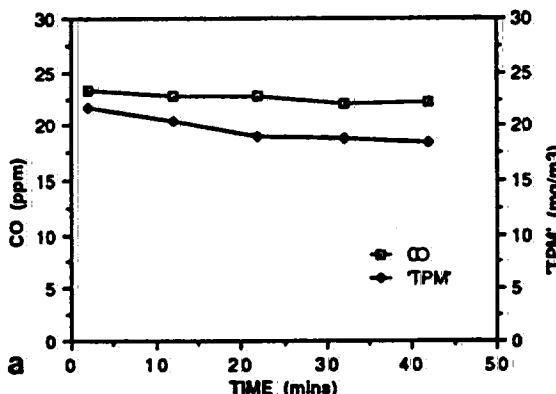


Fig. 1. a Changes in ambient concentrations of carbon monoxide and "Miniram" particulates. b Changes in ambient concentration of nicotine

2023380186

Particulates were measured using the MINIRAM (Miniature Real-time Aerosol Monitor, GCA Corporation, Model PDM-3), a light scattering device which samples over 10-s-time periods. This instrument gives a quantitatively high result because of its sensitivity to particle size distribution and its dependence upon a relevant calibration [4].

Ammonia was measured continuously using a selective ion electrode.

A "whole smoke" gas chromatographic profile was obtained by actively drawing the ambient atmosphere through standard Perkin Elmer ATD50 tubes packed with Tenax TA, 60-80 mesh for a 15-min-period at a flow rate of 300 ml/min. The Tenax was then thermally desorbed in two stages onto a 50 m mixed Ucon phase capillary column. Values for 33 distinct peaks were calculated as the peak area relative to that of the Internal Standard (Dimethyl Furan), these included Acetone, Acrolein, Acetonitrile, Pyridine and 3-vinyl pyridine.

A "phenolic profile" was obtained by drawing the atmosphere through a small Cambridge filter pad for 10 min at a flow rate of 20 l/min. The pad was then silylated using BSTFA and Digol was added as an Internal Standard. This was then heated for 1 h at 80°C and run on a 25 m SE54 capillary column. Values for 26 peaks, including Catechol, Glycerol, and Hydroquinone were calculated with reference to the Internal Standard.

### Results

Figures 1a and 1b show the results for nicotine, carbon monoxide and Miniram particulates. The carbon monoxide levels remain constant at the relatively high level of 22 ppm throughout the 50-min-study period, this being consequent upon the defined

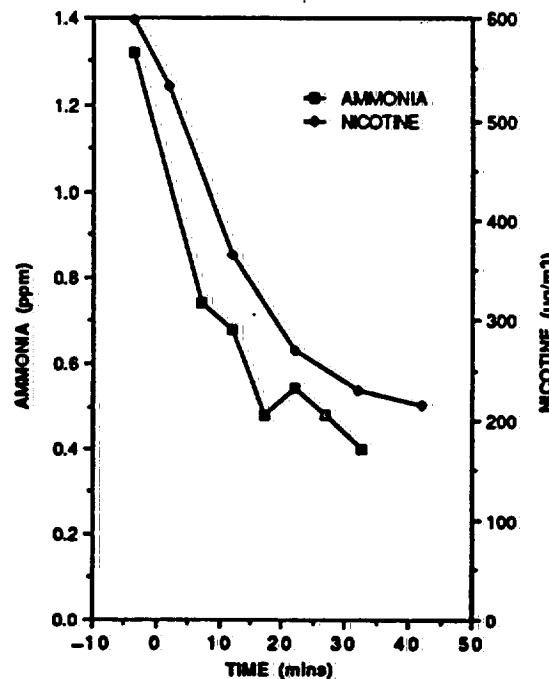
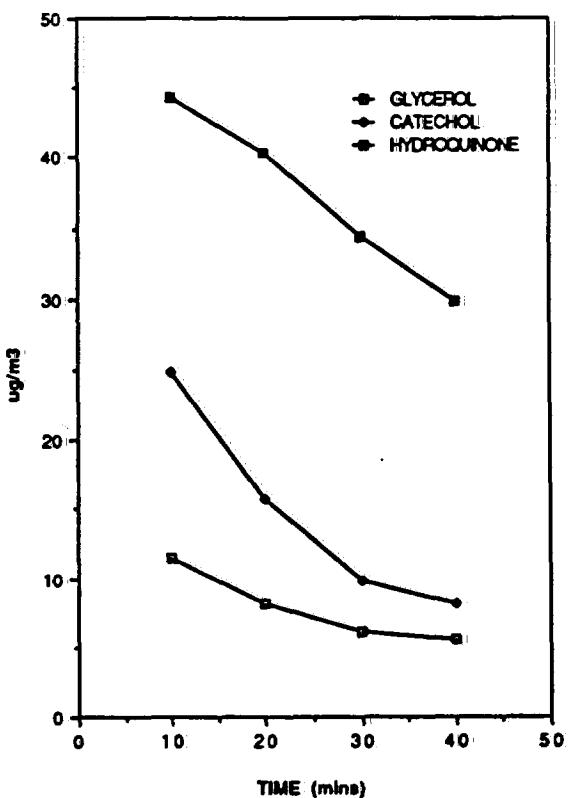


Fig. 2. Changes in ambient concentration of nicotine and ammonia

2023380187



**Fig. 3. Changes in ambient concentration of Catechol, Glycerol, and Hydroquinone**

smoking regimen. The particulate levels can be seen to fall by about 15% and this is most likely accounted for by the loss of volatile materials to the ambient atmosphere. In contrast to this relatively small decline in particulate levels however is the rapid fall in airborne nicotine levels which decay to less than 20% of their initial value.

Figure 2 shows that the levels of ammonia exhibit a similar rapid decay to that seen for nicotine.

Examples from the analysis of the "phenolic profile" are given in Fig. 3 which illustrates the decay of Catechol together with Glycerol and Hydroquinone. These results draw attention to the fact that whilst the majority of components appear to show an exponential decay pattern this is not invariable and as an example Hydroquinone appears to decay over this time period in a linear fashion.

Because of the longer periods over which the "whole smoke" profile samples are obtained it is not possible to display the changes graphically. Comparing the time periods 0-15 min with 30-45 min gives some idea of the variability in rates of decay. These are illustrated in Table 1 where the percentage change of individual peak areas between the two periods can be seen to range from 0% to 40%.

Of the 50 plus components of sidestream smoke examined in these studies in no case was any component found to increase over the 50-min-time period.

2023380188

Table 1. Levels of major components in the "whole smoke" profile of an ambient air sample and their % change over a 45-min-time period.

Peak No.	Identification	15-30-min-value	45-60-min-value	% change
1		1.51	1.64	-5
2		0.62	0.61	-13
3		4.40	3.94	-11
4		0.65	0.74	0
5		0.52	0.46	-12
6		0.36	0.30	-17
7	Acetone	0.77	0.87	0
8	Acrolein	0.34	0.38	-3
9		1.28	1.22	-5
10	Pyridine	1.05	1.10	-5
11	Acetonitrile	0.67	0.68	-1
12		0.68	0.58	-15
13	Benzene	2.66	2.36	-11
14		0.52	0.53	-2
15	Int. Standard	1.00 (33.5)	1.00 (29.4)	
16	Toluene	4.51	5.20	15
17		0.79		
18		2.31	1.93	-17
19		2.29	1.73	-30
20	3-Vinyl pyridine	0.70	0.76	9
21	Phenol	1.99	2.28	14
22		2.25	1.75	-27
23		0.89	0.75	-17
24		1.20	1.00	-17
25		1.31	1.23	-6
26		3.53	3.00	-15
27		1.92	1.90	-1
28		1.45	1.40	-3
29		1.83	1.56	-15
30		2.01	1.68	-17
31		1.64	1.25	-25
32		1.02	0.74	-27
33		0.65	0.44	-30

Values presented were calculated by the (peak area of component)/(peak area of Internal Standard). Values in brackets were the actual peak areas. The % change between the results allows for the differences in value for the Internal Standard.

### Discussion

The results presented in this paper clearly demonstrate the variability in the decay pattern for individual components of environmental tobacco smoke. Although the measurements were made in an experimental situation at a relatively high ambient smoke level this variability would certainly be encountered in the real-life situation.

It is thus clear that to make extrapolations from the measurement of a single marker to the behavior of other smoke components involves an assumption which is likely to be

2023380189

1. Nyst  
indo  
indo
2. Bau  
mac
3. Bro  
com
4. Raw  
smc

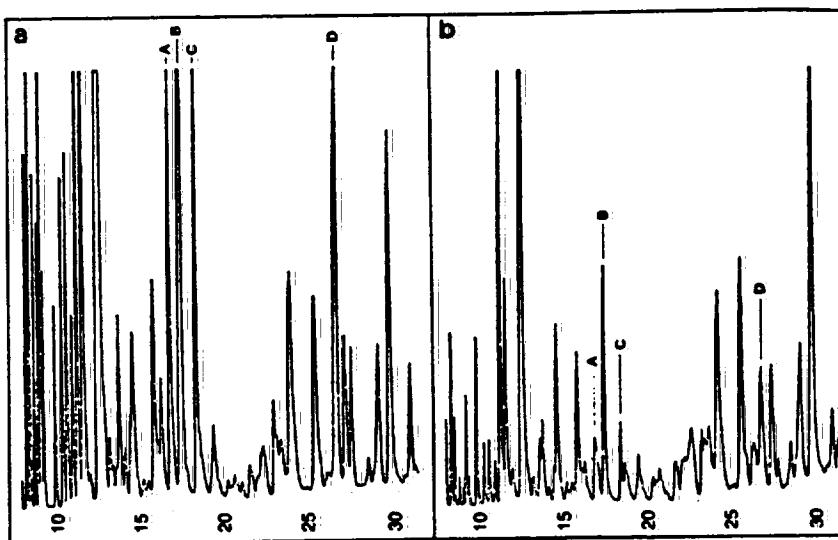


Fig. 4a, b. "Whole smoke" chromatographic profiles of (a) freshly generated sidestream smoke in a benchtop apparatus and (b) environmental tobacco smoke following the dispersion of sidestream smoke from 16 cigarettes.

invalid. One further point can be noted from a comparison between fresh sidestream smoke measured in a benchtop apparatus and sidestream generated in the experimental room. This is illustrated in Fig. 4 which presents the whole smoke profiles obtained in a Keith apparatus with that taken in the experimental room immediately following the smoking of the 16 cigarettes. The four components labelled A, B, C and D, which have been provisionally identified as Furan, Acetone, Acrolein and Acetonitrile, are among those which can be seen to have greatly reduced levels in the room relative to those in the benchtop collection device. Although these components appear in fresh smoke their apparent decay is so rapid that they may not be seen to any significant extent in room air.

### Conclusions

- 1) Environmental tobacco smoke is a dynamic aerosol which exhibits both temporal and spatial variation.
- 2) Each of the components of smoke measured has its own decay rate and pattern. Relative to carbon monoxide and particulates, nicotine and ammonia have rapid decay rates. Other components, which probably include Acrolein, decay at an even faster rate and high airborne levels are probably never achieved.
- 3) Extrapolations from benchtop sidestream measurement to room air based on simple dilution calculations is unlikely to provide valid information.
- 4) Extrapolations from a measured "marker" in ambient air studies to other potential smoke components should be performed with caution.

2023380190

*References*

1. Nystrom CW, Green CR (1986) Assessing the impact of environmental tobacco smoke on indoor air quality: current status. Proceedings of the ASHRAE conference IAQ86, Managing indoor air for health and energy conservation, Atlanta, Georgia
2. Baumgartner H, Coggins CRE (1980) Description of a continuous-smoking inhalation machine for exposing small animals to tobacco smoke. Beitr Tabakforsch Int 10:169-174
3. Browne CE, Keith CH, Allen RE (1980) The effect of filter ventilation on the yield and composition of mainstream and sidestream smokes. Beitr Tabakforsch Int 10:81-90
4. Rawbone RG, Burns W, Patrick RA (1987) The measurement of "environmental tobacco smoke" particulates. Toxicology Letters 35:125-129

2023380191

## The Measurement of ETS Through Adsorption/Desorption Procedures

C. Proctor and H. Dymond

### Introduction

Environmental tobacco smoke (ETS) has received increasing attention in recent years, yet there is still no clear way of precisely measuring ETS. This is due to several compounding factors. ETS is an extremely complex mixture of compounds [1], it is much diluted and integrated with the ambient air and hence any compounds present from other sources, and it is not a stable entity [2]. Because of the dilution factor and the complexity involved one must use a highly sensitive and selective analytical technique that can determine the presence of chemicals specific to tobacco smoke. The alternative is to measure an environment with and without tobacco smoke present, but this is rarely possible in realistic situations [3]. Furthermore the technique must take account of the fact that ETS is continuously changing; it is analytically a moving target. This paper presents a method that allows the acquisition of chromatographic profiles of ambient air.

ETS originates from the combination of the sidestream smoke of a burning cigarette, the exhaled mainstream smoke and any smoke spilled from the mouth during draw (mouthspill). Its concentration and composition will depend on many factors, including the type of tobacco product smoked, the number smoked, the air movement conditions in the environment, and the type of adsorbent surfaces such as walls and furnishings present. It will consist of chemicals in both the gaseous, the vapour and the particulate phases. Moreover, it is a dynamic aerosol and some compounds traditionally associated with the particulate phase of smoke are found in the vapour phase of aged ETS as the particles tend to lose volatiles with time [4]. Associated with this phenomena is the fact that different compounds in ETS will have different decay rates.

Some constituents of ETS may be measured directly by portable and sensitive equipment [5], but this generally is only applicable to gases such as carbon monoxide which are non-specific to tobacco smoke and will be produced by other forms of combustion [6]. More specific chemicals, such as nicotine, when in the low concentrations found in ETS require concentration steps in the analytical method. An appropriate method for achieving this, and at the same time producing chromatographic profiles of the ambient atmosphere is described here.

### Collection of the Sample

Adsorption traps have become increasingly more accepted in methods aimed at measuring concentrations of chemicals in ambient atmospheres [7]. The general principle is very simple. A known volume of air is drawn through a chemical support with adsorbent properties. If the correct adsorbent is used and concentrations are not so high as

to cause breakthrough, then sampling can take place over several hours. The longer the sampling period, the better the sensitivity of the analysis. However, this should be balanced by the fact that the measurement is an average over a period of time and does not account for short-term temporal variations, though this may also be desirable.

In assessing the capability of an adsorbent system it is necessary to investigate one compound at a time. The obvious choice for ETS is nicotine. This is because it is specific to tobacco smoke, it is in high concentration relative to other volatile components and because it has been traditionally a measure of mainstream smoke. Numerous analytical methods for the determination of ambient nicotine have already been published [8]. All use an initial trap for concentration of the sample. The National Institute for Occupational Safety and Health Administration (NIOSH) recommend that the nicotine is trapped on Amberlite XAD-2 resin [9]. The sample can then be eluted with a quantity of ethyl acetate and quantified by gas chromatography. Liquid desorption, however, introduces a considerable dilution factor to the analysis and thus does not allow the attainment of very low limits of detection. The sensitivity of the method is much improved if thermal desorption of the adsorbent is used as then the total sample is analysed in one go.

Adsorbents applicable to thermal desorption must fulfill several criteria:

- the adsorbent must be efficient in trapping the chemical under consideration, whether that compound is in the particulate or the vapour phase;
- the efficiency should be such that there is no breakthrough of compound during long sampling periods;
- the adsorbent must however be able to release all of the compound after thermal desorption for a short period of time and at a temperature below that likely to degrade the sample;
- the adsorbent itself must be chemically inert and thermally stable (to avoid leaching of compounds associated with the support complicating the analysis);
- the adsorbent must be able to trap the sample for some length of time without degradation to allow for the transfer time between the sample being collected and the eventual analysis time.

We found that the adsorbent Tenax TA, which is a polymer of 2,6-diphenyl-p-phenylene oxide [10], satisfied all of these criteria when considering the collection of nicotine. Several experiments were run before coming to this conclusion. In all experiments a weight of 0.4 g Tenax TA (35–60 Mesh) was packed into a stainless steel tube as described in Fig. 1.

The first experiment was to assess the collection efficiency of the trap to ambient smoke particulates. This was done using a Malvern LASX Laser Aerosol Spectrometer. This instrument is limited to a lower size range limit of 0.09 to 0.11  $\mu\text{m}$ . Samples of 40 cc of both fresh sidestream smoke and aged ETS were collected and introduced into an inert bag containing 2,000 cc of nitrogen. The samples were then analysed by the spectrometer with and without a trap between sample and analyser. The experiment showed the trap to have a collection efficiency for all particulates of 96% for "fresh" smoke and 93% for aged smoke. This efficiency was consistent over the range of particles observed from 0.09 to 2  $\mu\text{m}$ . A similar experiment, but using the adsorbent Supelcoport 100/120 mesh containing 5% OV17 in the trap, gave trapping efficiencies for all particulates of 99.6% for "fresh" smoke and 99% for aged smoke. Even though the Tenax efficiency is not as good, further factors make it useful.

The second series of experiments set out to determine the collection efficiency of vapour phase constituents. Nicotine is thought to be almost entirely in the vapour phase in

2023380193

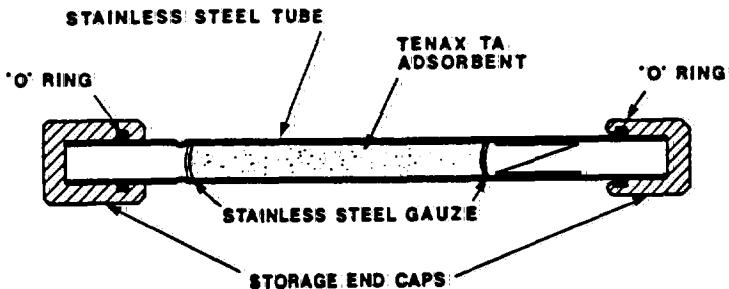


Fig. 1. Schematic diagram of the sampling tube used for adsorption

aged smoke [2] and so it was used for this check. By sampling ETS produced in a controlled room through two tubes in series it was determined that the first tube was 99% efficient.

The good thermal stability of Tenax is well documented, and so the third experiment investigated the retention of nicotine on the tube during thermal desorption. By injecting liquid standards of nicotine in propanol both onto the Tenax and directly into the analysis system, and by analysis of ETS samples, it was determined that a desorption of 15 min at 150°C released 99% of the nicotine for subsequent analysis.

Storage of trapped samples was also considered, and it was found by taking samples of ETS in parallel that there was no deterioration in nicotine content over two week refrigerated storage [11].

There has been much data published on the effectiveness of Tenax as an adsorbent for a wide range of volatile materials [12]. Within the regime of using 0.4 g of Tenax and typically sampling 1,000 cc of air at a rate of 10 cc per minute, the tubes will be efficient for the majority of volatile compounds present in ambient air.

#### Analysis of the Sample

Such complex mixtures as found in ambient air require a powerful separation stage in order to resolve the individual components. In order to attain good resolution the sample must be presented to the chromatographic column as a discrete sample. Therefore, direct thermal desorption (which requires 10 to 15 min for complete release) will result in a poorly resolved chromatogram. This can be overcome by the introduction of a cryofocusing step in the procedure. The sample of trapped and concentrated ambient air is swept off the trap by being heated at 150°C for 15 min whilst a flow of helium gas flushes the desorbed components through the system and into a cold trap containing a small amount of Tenax (approximately 0.05 g) maintained at -30°C. This secondary, cryofocusing trap is then rapidly heated electronically in order to "inject" the collected compounds onto the chromatographic column.

Our instrumental set-up is illustrated in Fig. 2. A Perkin-Elmer ATD-50 is used for the two stage desorption. The carrier flow can be split both before and after secondary trapping. The head of the chromatographic column is positioned directly after the cold trap. A heated transfer line containing the column then links the trap to a Perkin-Elmer Sigma 3 gas chromatograph. The exit of the column is fed directly into the ion source of

2023380194

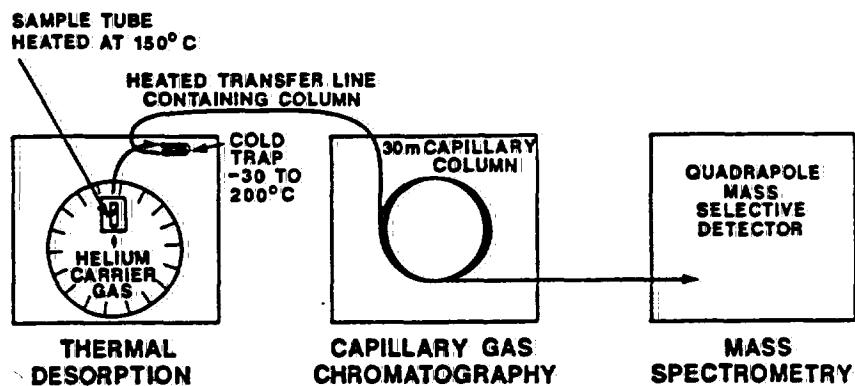


Fig. 2. Schematic diagram of the analytical instrumentation used to analyse the adsorbed samples.

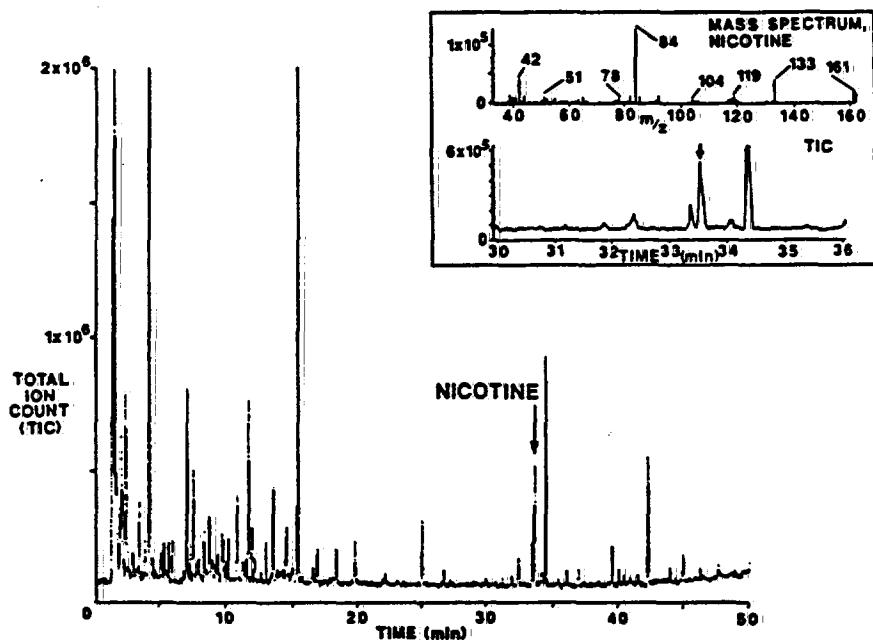


Fig. 3. Chromatographic profile of the ambient atmosphere in a bar in a public house

a Hewlett-Packard mass selective detector. This combination of analytical techniques allows introduction of the concentrated sample, followed by high resolution separation of the individual components, followed by identification and quantification of each compound by the mass spectrometer.

2023380195

Breakthrough of compounds through the cold trap can be monitored by running the mass spectrometer during primary desorption. When using the apparatus to measure ETS samples, this is rarely a problem. Tenax is hydrophobic and so any moisture collected during sampling will not cause analytical problems such as freezing of the cold trap.

The mass spectrometer is a very selective and sensitive device. From the fragmentation patterns produced by electron impact most compounds can be uniquely identified. The sensitivity of the device allows the measurement of sample in sub nanogram concentrations.

#### Examples of Chromatographic Profiles of Ambient Atmospheres

The following are examples of the type of chromatographic profiles that may be obtained with the analytical system described. In each case sample air volumes of between 1 and 2 litres were taken at approximately head height from a static position and no attempt was made to avoid close contact with smokers. Sample flow rate was 0.6 l/h for each sample. Analysis of samples was achieved in every case with a primary desorption of 15 min at 150°C onto a cold trap maintained at -30°C. Secondary desorption heated the cold trap from -30 to 200°C, thus introducing the sample to a 30 m, 0.25 µm Supelco SPB-5 capillary column. The mass spectrometer was run in total ion mode with a multiplier set at 2000 V.

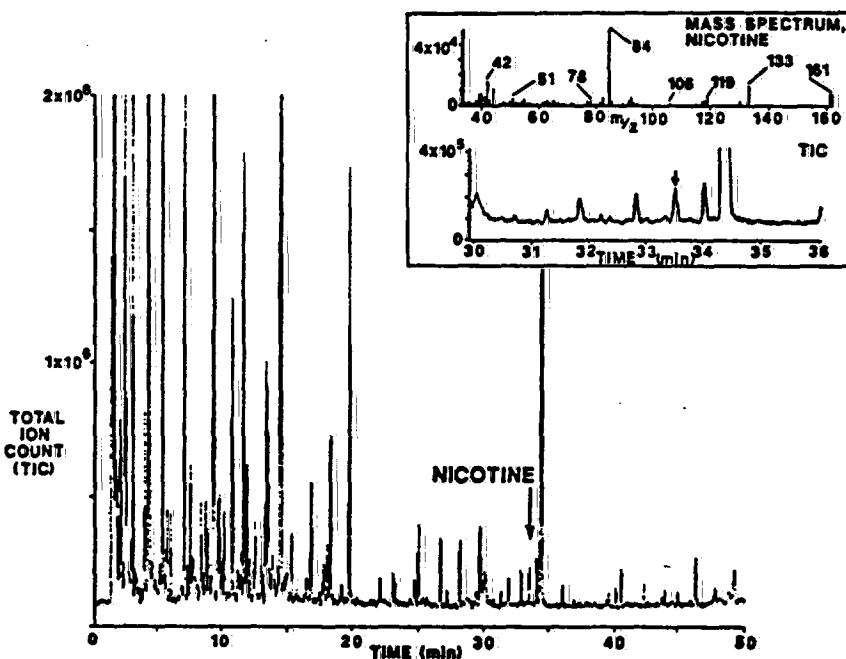


Fig. 4. Chromatographic profile of the environment in the living room of a private house

2023380196

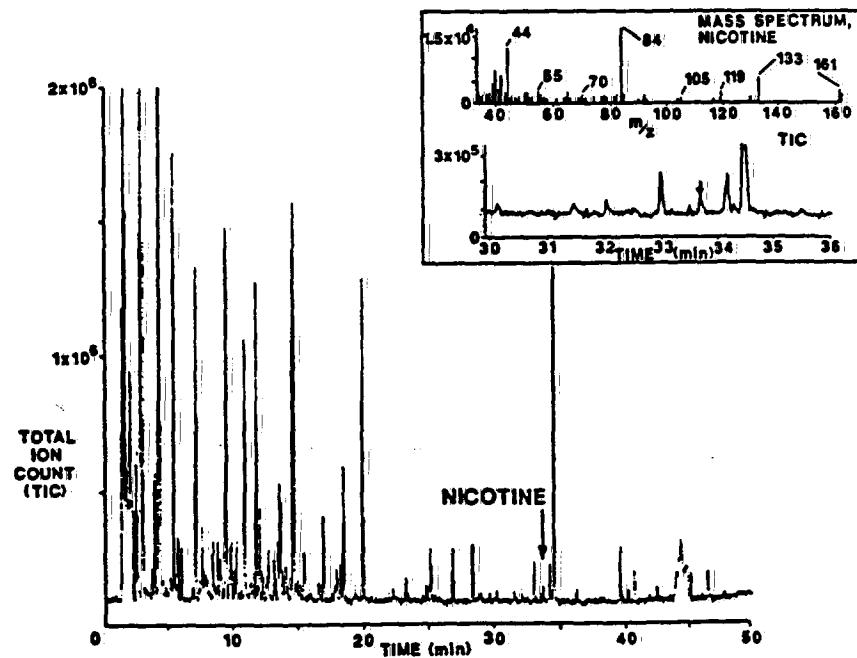


Fig. 5. Chromatographic profile acquired in the kitchen of a private house during cooking in an electric oven

The first example was taken in the bar of a public house during a lunchtime period. There were more than six active smokers present, three of which sat close to the monitoring point. Figure 3 shows the chromatographic profile for this sample. The nicotine peak corresponds to an ambient concentration of  $38 \mu\text{g}/\text{m}^3$  of nicotine. There are clearly a large number of compounds present in this atmosphere. For example, the large peak at 15.5 min retention time corresponds to dichlorobenzene. This presumably arises from the use of a cleaning agent in the pub.

Figure 4 shows the chromatographic profile corresponding to the ambient air in the living room of a private house. Two people smoked a total of six cigarettes during a 2-h sampling period. The ambient nicotine concentration averaged over this period was  $8 \mu\text{g}/\text{m}^3$ . Many of the other compounds observed were found to be aliphatic hydrocarbons. Figure 5 was acquired in the kitchen of the same house during the cooking of a meal using an electric oven. There the nicotine level was found to be  $3 \mu\text{g}/\text{m}^3$ . The majority of the chemicals identified were common to both environments.

The ambient atmosphere in a car during a 2-h-motorway (high speed) journey is illustrated in Fig. 6. Five cigarettes were smoked by the driver during the trip, the sample was taken in the position of a front seat passenger, and the air ventilation devices and windows remained closed for the majority of the journey. The average ambient nicotine content was found to be  $8 \mu\text{g}/\text{m}^3$ . Again the profile is complex and contains many compounds.

2023380197

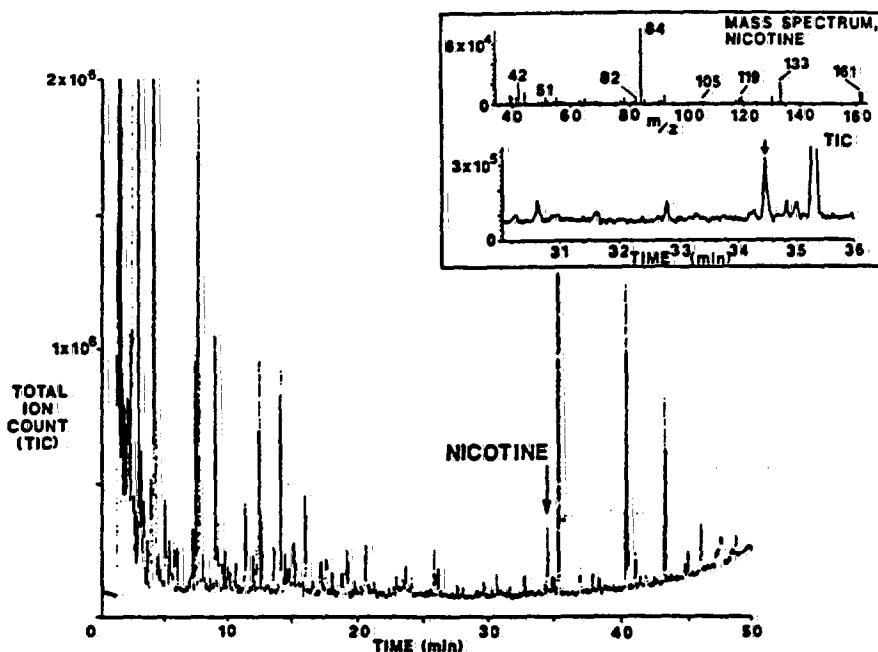


Fig. 6. Chromatographic profile of the ambient air in a car during a 2-h-journey

Finally, Fig. 7 presents the profile of the atmosphere in an Indian restaurant during a meal taken by two smokers over the period of two hours. Three other people were noticed to be smoking during the same period. Nicotine level was found to be  $12 \mu\text{g}/\text{m}^3$ . More than 200 other chemicals were observed in the analysis; many of them being volatile "flavour" type compounds.

#### Conclusions

This work has demonstrated that adsorption/thermal desorption procedures can be used to measure volatile compounds present in ambient atmosphere. However, the chromatographic profiles given as examples make it clear that ambient air consists of a complex mixture of compounds. Moreover, the analysis of several realistic environments, all of which contained ETS, shows large differences in the individual chemicals present in different atmospheres. As ETS is common to the experiments, these differences presumably arise from the contribution of various sources other than tobacco smoke.

Therefore, any measurement of ETS, whether it takes nicotine or some other compound specific to tobacco smoke as a marker, must use analytical methodology capable of high resolution of the mixture. It should also use a detection technique capable of specific identification of the compound because, as has been shown in this paper for the case of nicotine, the peak of interest is likely to be small relative to signals arising from other volatile compounds.

2023380198

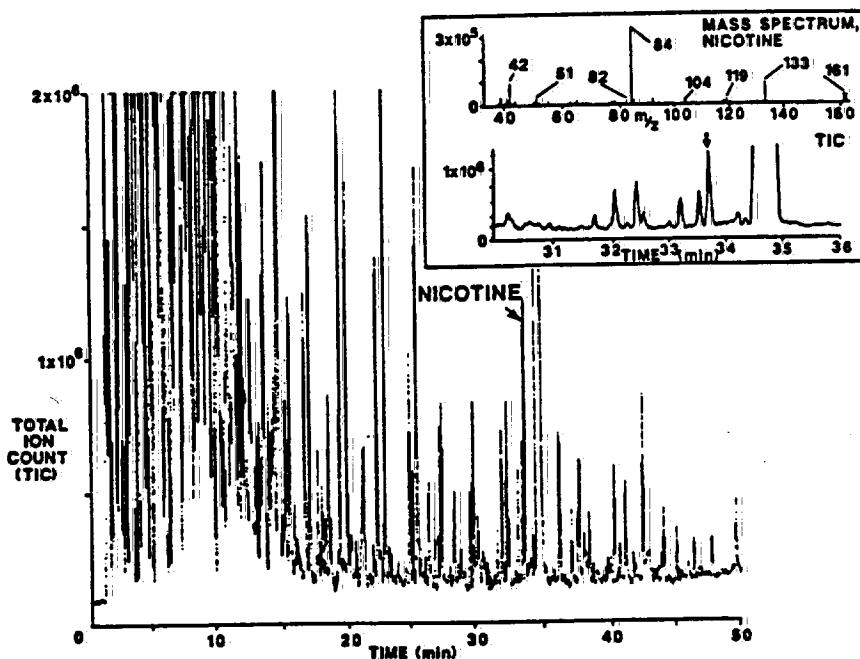


Fig. 7. Chromatographic profile of the atmosphere of an Indian restaurant

### References

1. Baker RR (1981) Product formation mechanisms inside a burning cigarette. *Prog Energy Combust Sci* 7:135
2. Nystrom CW, Green CR (1986) Assessing the impact of ETS on indoor air quality; current status. Proceedings of the ASHREA conference IAQ86. Managing indoor air for health and energy conservation, April 20-23rd, Atlanta, Georgia
3. Sterling TD, Dimich H, Kobayashi D (1982) Indoor by-product levels of tobacco smoke; a critical review of the literature. *J Air Pollution Cont Ass* 32:250
4. Heavner DL, Thome FA, Eudy LW, Green CR (1985) A test chamber and instruments for the analysis of ETS. 39th Tobacco Chemist's Research Conference, Montreal
5. Proctor CJ (1987) A study of the atmosphere in London underground trains before and after the ban on smoking. *Toxicology Letters* 35:131
6. Brunekreef B, Smit HA, Biersteker K, Boleij JS, Lebret E (1982) Indoor carbon monoxide pollution in the Netherlands. *Environ Int* 8:193
7. Brown RH, Purnell CJ (1979) Collection and analysis of trace organic vapour pollutants in ambient atmospheres; the performance of a Tenax GC adsorbent tube. *J Chrom* 178:79
8. Muramatsu M, Umemura S, Okada T, Tomita H (1984) Estimation of personal exposure to tobacco smoke with a newly developed nicotine personal monitor. *Environ Res* 35:218
9. NIOSH Manual of Analytical Methods (1977) 2nd ed, vol 3; Dept. of Health, Education and Welfare, No. 5293, April
10. Daemen JMH, Dankelman W, Hendriks ME (1975) Properties and applications of Tenax GC as a column packing material in gas chromatography. *J Chrom Sci* 13:79
11. Zlatkis A (1977) Concentration and analysis of volatile urinary metabolites. *J Chrom* 142:755
12. Holzer G, Shanfield H, Zlatkis A, Bertsch W, Juarez P, Mayfield H, Lieblich HM (1977) Collection and analysis of trace organic emissions from natural sources. *J Chrom* 142:755

2023380199

## Assessment of ETS Impact on Office Air Quality

J.J. Piadé, C. Gerber, and W. Fink

### Summary

The contribution of environmental tobacco smoke (ETS) to indoor air quality was investigated by quantifying the concentration of some of its constituents in the course of a series of strictly controlled experiments.

One brand of commercial cigarettes was smoked by trained smokers following a prescribed protocol both in a test-chamber and in an office of a modern, air-conditioned building. The ETS components investigated were CO, NO, NO<sub>2</sub> and nicotine. The concentration of respirable suspended particles (RSP) was also monitored using three different methods.

The concentrations of these ETS constituents and their ratios are reported, together with background and outdoor levels. In addition, the influence of room ventilation, smoke generation rate, wall deposition effects, etc., is discussed.

### Introduction

The indoor air concentration of ETS components has been surveyed by many authors in real-life measurements, but with little or no information on smoke generation. In other reports, mostly for exposure studies, both smoke generation and air concentration of several ETS components were carefully monitored, but with often unrealistic smoke levels [1, 2].

This paper is the first part of a study aimed at investigating ETS chemistry in real-life situations, but with a strictly defined smoke generation and investigating a wide array of components. It comes as a continuation of previous investigations on sidestream smoke (SS) generated in a test-chamber [3]. In this study the effects of smoke generation patterns, room ventilation and air mixing should be assessed, with an emphasis on the time variation of the measured concentrations and their ratios. This paper reports on early results establishing the experimental concept, checking methods and evaluating the impact of various indoor environmental factors.

### Experimental Procedures

#### *Smoking Sessions*

The office used for this study has a surface of 12 m<sup>2</sup> and a volume of 35 m<sup>3</sup>, with a door and a large window. Its walls are plastered, the floor is carpeted and it is furnished with a desk, three chairs and a cupboard. It is situated in a modern building

with central air conditioning. The ventilation was checked to ensure 3.5 air changes per hour.

Smokers normally consuming about 1 pack per day were trained to take 2-s puffs per minute in a reproducible way, as checked by consistent puff-counts per cigarette. They were asked to smoke commercial cigarettes according to a pre-determined, realistic protocol. All smokings took place in the same room, but ventilation was turned on or off with possible additional air mixing.

#### *Analytical Methods*

For each session, the concentrations of CO, NO, NO<sub>2</sub> and respirable suspended particles (RSP) were measured continuously. Nicotine concentration was measured periodically.

Samplings were done using feed-back flow control pumps (SKC Aircheck Sampler 224-36) drawing air from near the center of the room at an height of about 1.2m.

Carbon monoxide was measured continuously by non-dispersive IR (Dasibi 3008) and nitrogen oxides by chemiluminescence (Tecan CLD 502).

Nicotine was sampled by pumping air through XAD-4 tubes (SKC 226-30-11-04) which were extracted with 1 ml of ethyl acetate (0.01 % triethylamine) and analysed by capillary gas chromatography according to [4]. Quinoline was used as an internal standard.

RSP concentration was simultaneously measured by three different methods:

- Filter gravimetry, by pumping air at 2 l/min through a filter pad (Fluoropore, Millipore FALP03700), possibly after passing through an impactor (TSI 3.5 µ cut-off) retaining particles that would not be inhaled [5], according to [4]. The weight change was measured with a microbalance (Mettler M3).
- Portable piezobalance (TSI model 5500).
- RAM nephelometric detector (GCA RAS-1).

#### *Instrument Calibration for RSP Determination*

The gravimetric determination is a direct method which is well established [4, 6]. It is precise down to about 30 µg/m<sup>3</sup> for 1-h samplings and the coefficient of variation of replicate analyses is about 4%. It only provides time-averaged answers, whereas the RAM gives almost real-time readings and the piezobalance provides a result every 3-5 min.

The TSI 5,500 is factory calibrated and gives direct readings of RSP levels (mg/m<sup>3</sup>). It has been used in many ETS studies [7] and its performance has been questioned by several authors [2]. The manufacturer reports it to underestimate tobacco smoke by 15% [9] and in a recent study significant differences between the responses of two identical instruments were reported [8]. The response of the TSI 5,500 we used to SS (between 0.09 and 1.2 mg/m<sup>3</sup>) was compared to gravimetric determinations in a series of experiments performed in our test-chamber. The difference between both determinations was consistently smaller than the variability of the methods, provided that the sampling flow rate of the piezobalance was kept at exactly 1 l/min and that its sensor was washed after each determination.

In contrast to the piezobalance, the RAM has to be calibrated before use with the aerosol studied [10]. This is due to its sensitivity to the particle size distribution of the

2023380201

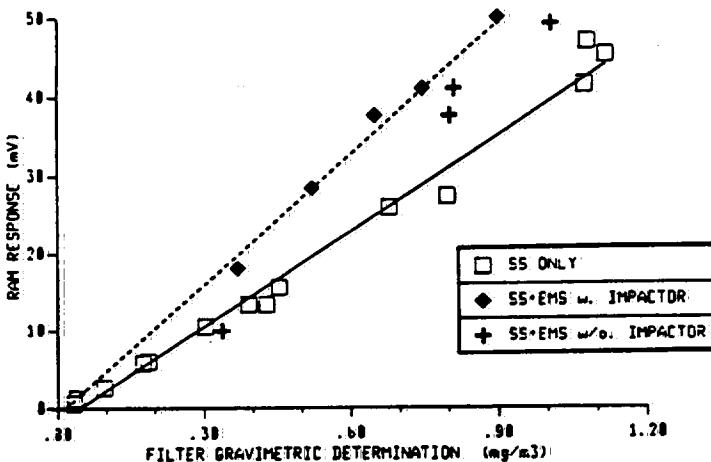


Fig. 1. Calibration of RAM vs. gravimetric determinations

sample. To this aim, the time-averaged RAM output was compared to gravimetric results in a series of experiments where smoke was generated in the test-chamber by SS only (machine smoking, mainstream smoke (MS) exhausted out of the room), or by SS plus exhaled MS (human smoking). Determinations were made for total airborne particulate matter or for RSP only (by sampling through  $3.5\text{ }\mu\text{m}$  impactors).

The results are given in Fig. 1. They reveal two possible sources of systematic error:

- If the RAM is calibrated using SS only for ETS measurements, RSP results will be significantly over-estimated.
- It is obvious that omitting the impactor will result in over-estimating the air burden if one should perform a direct gravimetric determination. But since the RAM response is practically not affected by the adjunction of an impactor, it is essential that the calibration be made by comparison with RSP only (i.e. using  $3.5\text{ }\mu\text{m}$  impactors at the filter and RAM inlets).

### Results and Discussion

For each smoking session of this first set of office ETS studies, the smoke generation protocols and the environmental conditions are given in Table 1.

In experiment 3, five cigarettes were smoked simultaneously, and the room ventilation was left on. Time zero was set at the moment when the cigarettes were extinguished. Figure 2 shows the plot, as a function of time, of the CO concentration together with that of RSP as measured with the RAM and with the piezobalance and the time-averaged concentration of nicotine. These values are all background corrected.

Figure 2 shows that the CO concentration decreases exponentially. The calculated decay rate is almost equal to the measured air changes per hour in the room. Thus CO is a good tracer that can be used to offset the effects of room ventilation.

The RSP concentration as measured by the RAM also decreases exponentially, a little faster than the CO. Thus the RSP to CO ratio does not remain constant with time.

2023380202

Table 1: Smoke generation protocol and environmental conditions

Experiment code	Number of cigarettes smoked	Generation rate	Room ventilation
1	1	at time 0	on
2	2	at time 0	on
3	5	at time 0	on
4	9	every 15 min	on
5	2	at time 0	off
6	4	every 15 min	off
7	4	every 15 min	off, fans on

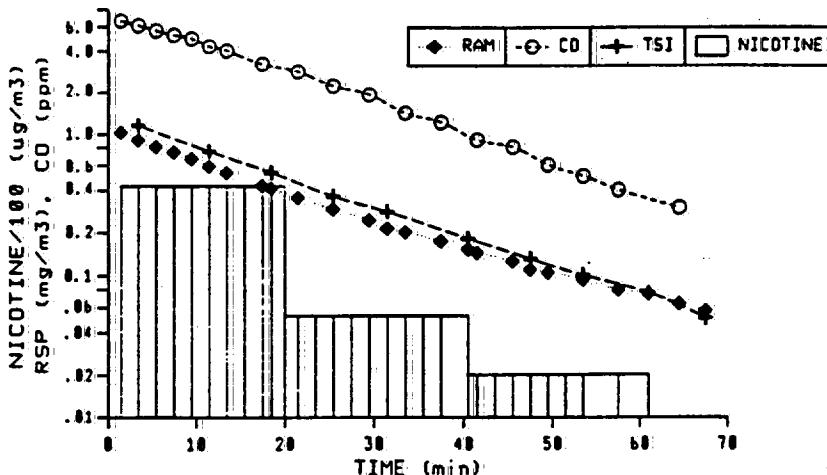


Fig. 2. RSP, CO, and nicotine decay after smoking 5 cigarettes

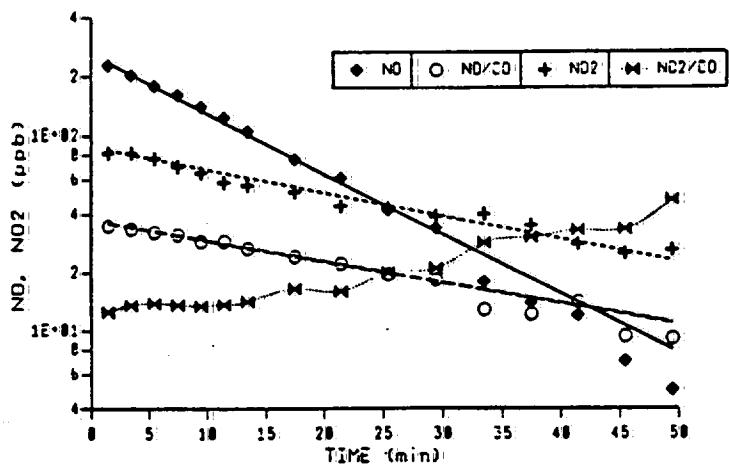
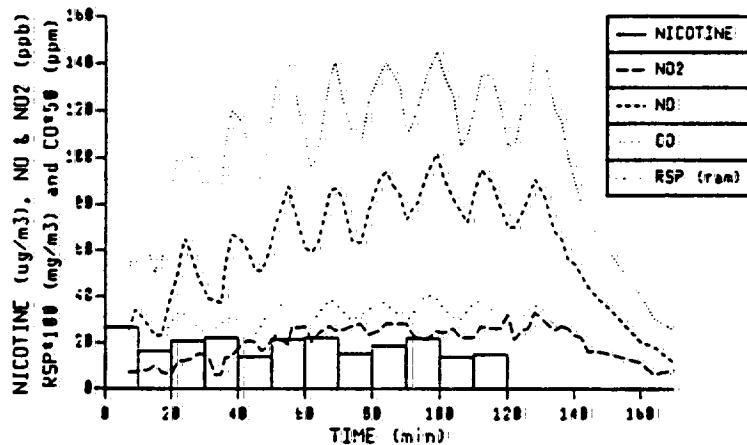
Actually, the decay rate of the RSP/CO ratio reflects the kinetics of wall impaction and sedimentation of the particles.

If we now consider the piezobalance determinations, they are slightly higher than the RAM measurements for unaged ETS. After about 40 min both curves coincide. An explanation for this discrepancy may be sought in changes in the smoke particle size during the early aging phase [11].

The plot of the nicotine concentration shows that it decays much faster than RSP immediately after smoking. After 1 h, the level drops much more slowly, actually even more slowly than the CO. This is probably due to the fact that nicotine is mostly present in the gas phase [12], and wall effects become very important. Of course the nicotine/RSP ratio is far from remaining constant.

Figure 3 shows the time variation of NO and NO<sub>2</sub> concentrations. The decay of the NO concentration appears to be exponential. Considering the NO/CO ratio, which offsets the effect of room ventilation, evidences the contribution of what seems to be a pseudo-

2023380203

Fig. 3. NO, NO/CO, NO<sub>2</sub>, and NO<sub>2</sub>/CO decay after smoking 5 cigarettesFig. 4. RSP, CO, NO, NO<sub>2</sub>, and nicotine concentration; 9 cigarettes smoked at 15-min-intervals

first order chemical decay. It should be noted that the NO decay was recently reported to be pseudo-first order in MS gas phase, but pseudo-second order in the whole MS [13]. The time increase of the NO<sub>2</sub>/CO ratio, on the other hand, reveals a chemical generation of NO<sub>2</sub> in the early phase of ETS aging. Of course, the NO<sub>2</sub> level decreases in absolute value after a few minutes.

A steady-state situation can be created with a constant smoke generation rate. This is what is obtained in experiment 4, where a cigarette is smoked every 15 min with the room ventilation left on. The corresponding profiles are shown on Fig. 4.

2023380204

Table 2. Time averaged RSP and nicotine concentrations.

Experiment code	RSP		Nicotine	
	Sampling time (min)	Concentration (mg/m³)	Sampling time (min)	Concentration (µg/m³)
1	90	0.089	40	8.6
2	96	0.189	40	18.6
3	79	0.391	40	25.6
4	150	0.478	40	21.4
5	121	0.350	40	25.3
6	128	0.508	40	28.7
7	130	0.486	40	16.8
Indoor background		0.033		0.7

Each time a cigarette is smoked, there is a rise and subsequent decay of the CO, NO and RSP concentrations, and after about 1 h a steady-state concentration is achieved. Even the nicotine level becomes fairly constant after a brief initial peak. This kind of experiment could be very useful in determining how environmental conditions may affect the ratio between the concentrations of two ETS components.

The effect of changes in the environmental conditions can also be quantitatively evaluated when the time-averaged nicotine and gravimetric RSP concentrations obtained for all the situations investigated are compared. These results are gathered in Table 2 and perusal of this table allows the following comments to be made:

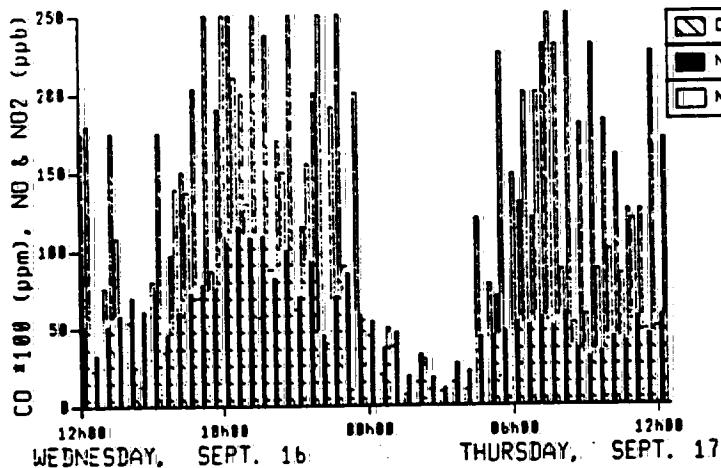
Comparing the RSP and nicotine averaged concentrations in experiments 1, 2 and 3, it appears that these values are not proportional to the number of cigarettes smoked, even in this strictly controlled set of experiments. This is even more true for the nicotine values and thus the nicotine to RSP ratio is fairly different in these three experiments. The drastic effect of room ventilation is obvious when comparing the results of experiments 2 and 5 or, in the case of continuous smoke generation, 4 and 6. Again, the impact of room ventilation is quite different whether one considers RSP or nicotine. Eventually, the effect of an increased air turbulence in the room is apparent when comparing the results of experiments 6 and 7. It appears that the average concentration of nicotine is much more reduced by air turbulence than that of RSP, pointing at the large influence of wall effects on nicotine concentration.

#### *Background Indoor and Outdoor Levels*

In average, the indoor background levels were about 0.6 ppm for CO, 10 ppb for NO, 50 ppb for NO<sub>2</sub>, 30 µg/m<sup>3</sup> for RSP and 0.7 µg/m<sup>3</sup> for nicotine.

In addition to indoor analyses, and in order to put these results in perspective, the outdoor concentration of CO, NO and NO<sub>2</sub> was measured, at the same time as the smoking sessions were held, by extending probes 1 m outside the window. The levels monitored over a 24-h period are plotted on Fig. 5. For nitrogen oxides, these values are at times higher than any level obtained in the course of our experiments. This is due in part to the proximity of a highway.

2023380205

Fig. 5. Outdoor CO, NO, and NO<sub>2</sub> concentrations.

### Conclusion

This study constitutes a first part of a program we have initiated on the analytical investigation of ETS in indoor air. Much more work is needed to obtain a good understanding of the main processes governing ETS aging. This initial study outlined some possible flaws in RSP measurement. It showed that a careful examination of the time variation of the measured concentrations and their ratios may yield valuable insights into ETS aging processes. As these ratios are not constant, it appears that no component can readily serve as a marker for other ETS components. In particular, nicotine was found to be quite outstanding in its behaviour, making it a poor marker of ETS exposure. Eventually the large impact of indoor environment factors such as air mixing, room ventilation, wall surfaces etc. on ETS was outlined.

### References

1. National Research Council (1986) The physicochemical nature of sidestream smoke and environmental tobacco smoke, measuring exposures and assessing health effects. National Academy Press, Washington DC, pp 25-53
2. Sterling TD, Dimich H, Kobayashi D (1982) Indoor by-product levels of tobacco smoke, a critical review of the literature. JAPCA 32:250-259
3. Blake CJ, Piadé JJ, Fink W (1986) Quantitative evaluation of cigarette sidestream smoke components under controlled experimental conditions. Presented at the International Experimental Toxicology Symposium on Passive Smoking, Oct. 23-25, 1986, Essen (FRG)
4. Ogden MW, Conner JM (1987) Methods of analysis for nicotine, respirable suspended particles and ultraviolet particulate matter in environmental tobacco smoke. Collaborative Study, Presented at the 41 Tobacco Chemists' Research Conference, Oct 4-7, 1987, Greenboro NC (USA)
5. Heyder J (1985) Studies of particle deposition and clearance in humans. In: Grosdanoff P et al (eds) Problems of inhalatory toxicity studies. BGA Publ, MMV-Medizin Verlag, Munich

2023380206

6. Pio CA (1986) General sampling techniques. In: Harrison RH et al (eds) Handbook of air pollution analysis. Chapman and Hall, London New York
7. Repace JL, Lowrey AH (1983) Indoor air pollution, tobacco smoke and public health, part I; Science 208 (1980) 464-472, Part II; World Smoking and Health 7(4) (1983) 35-46
8. Ingebretsen BJ, Heavner DL, Angel AL, Conner JM, Oldaker GB, Green CR (1986) A comparative study of environmental tobacco smoke particulate mass measurements in an environmental chamber. Presented at the International Experimental Toxicology Symposium on Passive Smoking, Oct. 23-25 1986, Essen (FRG)
9. Sem GJ, Tsurubayashi K, Homma K (1977) Performance of the piezoelectric microbalance respirable aerosol sensor. Am Ind Hyg Assoc J 38:580-588
10. Rawbone RG, Burns W, Patrick RA (1987) The measurement of environmental tobacco smoke particulates. Toxicology Lett 35: 125-129
11. Ingebretsen BJ (1986) Aerosol studies of cigarette smoke. Rec Adv Tob Sci 12:54-142
12. Eudy LW, Thome FA, Heavner DL, Green CR, Ingebretsen BJ (1985) Studies on the vapor/particulate phase distribution of environmental nicotine by selective trapping and detection methods. Presented at the 39 Tobacco Chemist Research Conference Oct. 2-5 1985, Montreal (CAN)
13. Borland CDR, Chamberlain AT, Higenbotham TW, Barber RW, Thrush BA (1985) A comparison between the rates of reaction of nitric oxide in the gas phase and in whole cigarette smoke. Beitr Tabakforschung Int 13(2):67-73

2023380207

# **THE HEALTH CONSEQUENCES OF INVOLUNTARY SMOKING**

*a report of the Surgeon General*

**1986**



**U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES  
Public Health Service  
Centers for Disease Control  
Center for Health Promotion and Education  
Office on Smoking and Health  
Rockville, Maryland 20857**

2023380208

---

For sale by the Superintendent of Documents, U.S. Government Printing Office  
Washington, DC 20402

determinant of the differing magnitudes of risks associated with these two exposures.

Differences in the composition of MS and SS primarily reflect their generation at different temperatures in different oxygen environments. Also, SS is diluted very rapidly, under most circumstances, and has the opportunity to age before inhalation. The involuntary smoker usually inhales ETS, not SS, the aerosol that comes from the tip of a burning cigarette. In considering the characteristics of SS, it must be emphasized that much of the existing data about the composition of MS and SS is derived from studies carried out in special chambers rather than by sampling MS and SS generated by smokers. In these chamber studies, SS has been sampled by a probe located close to the burning tip. This experimental situation clearly differs from that of a room with one or more smokers freely smoking. In that situation, SS is mixed with exhaled MS, diluted and aged. Nevertheless, these chamber studies provide very useful information about the compounds present in the SS. These studies have established that SS in comparison with MS has a higher pH, smaller particle size, and more carbon monoxide, benzene, toluene, acrolein, acetone, pyridine, ammonia, methylamine, nicotine, aniline, cadmium, radon daughters, benzo[a]pyrene and benz[a]anthracene.

Comparison of the relative concentrations of the various components of SS and MS smoke provides limited insights concerning the toxicological potential of ETS in comparison with active smoking. As described above, SS characteristics, as measured in a chamber, do not represent those of ETS, as inhaled by the nonsmoker under nonexperimental conditions. Further, the dose-response relationships between specific tobacco smoke components and specific diseases are not sufficiently established for the necessary extrapolations from active smoking to environmental tobacco smoke exposure for individual agents. For that reason the extrapolations in this section are confined to the dose-response relationships of whole smoke for those diseases with established dose-response relationships.

With regard to the potential of ETS to cause lung cancer, undiluted SS has 20 to 100 times greater concentrations of highly carcinogenic volatile N-nitrosamines than MS (Brunnemann et al. 1978) as well as higher concentrations of benzopyrenes and benz[a]anthracenes.

For nonmalignant effects on airways and the lung parenchyma, the agents responsible for the development of acute and chronic respiratory disease have not been identified, although many tobacco smoke components have been shown to cause lung injury (US DHHS 1984). Presumably, both vapor phase (gaseous) and particulate phase (solid) components of MS are involved. Both airways disease and

parenchymal disease are probably a response to the total burden of respiratory insults, some of which, like active smoking, may be sufficient by themselves to cause physiologic impairment and ultimately, clinical disease. Others, such as ETS, may contribute to the total burden but be insufficient, individually, to cause clinical disease.

#### *Deposition of Mainstream Smoke and Sidestream Smoke and Environmental Tobacco Smoke Dose Estimates*

The dose of tobacco smoke delivered to the airways and alveoli depends, among other factors, on the volume of MS, SS, or ETS inhaled, on the rate and depth of inhalation, and on the size, shape, and density of the individual particles or droplets. Patterns of deposition of MS in the lungs have been described, but similar information about deposition patterns for ETS is not yet available. Without such data, it is necessary to extrapolate from the information on MS.

The major factors that affect the pattern of deposition and retention for particles are particle size distribution and breathing pattern. The particle size range and mean aerodynamic diameter for particulates in sidestream smoke are similar to those of mainstream smoke (particle size range of 0.01 to 0.8  $\mu\text{m}$  for sidestream smoke and 0.1 to 1.0  $\mu\text{m}$  for mainstream smoke, and mean aerodynamic diameter 0.32  $\mu\text{m}$  for sidestream smoke and 0.4  $\mu\text{m}$  for mainstream smoke) (see Chapters 3 and 4). The deposition site is determined largely by the size of the particles, with large particles being deposited preferentially in the nasopharynx and large conducting airways. Smaller particles are deposited more peripherally, and very small particles tend to be exhaled and to have a very low deposition fraction. The particulates of ETS, because of their size range, are likely to be deposited peripherally.

The breathing patterns for the inhalation of MS and ETS are also different; MS is inhaled intermittently by the smoker with an intense inhalation, often followed by a breathhold that results in a more equal distribution. Environmental tobacco smoke, on the other hand, is inhaled continuously with tidal breaths when the passive smoker is at rest and with deeper inhalations when the passive smoker is physically active. Breathholding does not normally occur with tidal breathing.

Estimates of the equivalent exposure, in terms of cigarettes per day, resulting from ETS, as compared with MS, vary quite widely and depend on the way in which the estimates were made. Repace and Lowrey (1985) estimated that nonsmokers in the United States are exposed to from 0 to 14 mg of tobacco tar (average 1.4 mg) per day. Vutuc (1984) estimated that the exposure to environmental cigarette smoke is equivalent to 0.1 to 1 cigarette per day actively

---

# **ENVIRONMENTAL TOBACCO SMOKE**

---

## **Measuring Exposures and Assessing Health Effects**

---

**Committee on Passive Smoking  
Board on Environmental Studies and Toxicology  
National Research Council**

2023380210

**NATIONAL ACADEMY PRESS  
Washington, D.C. 1986**

National Academy Press • 2101 Constitution Avenue, NW • Washington, DC 20418

**NOTICE:** The project that is the subject of this report was approved by the Governing Board of the National Research Council, whose members are drawn from the councils of the National Academy of Sciences, the National Academy of Engineering, and the Institute of Medicine. The members of the committee responsible for the report were chosen for their special competences and with regard for appropriate balance.

This report has been reviewed by a group other than the authors according to procedures approved by a Report Review Committee consisting of members of the National Academy of Sciences, the National Academy of Engineering, and the Institute of Medicine.

The National Research Council was established by the National Academy of Sciences in 1916 to associate the broad community of science and technology with the Academy's purposes of furthering knowledge and of advising the federal government. The Council operates in accordance with general policies determined by the Academy under the authority of its congressional charter of 1863, which establishes the Academy as a private, nonprofit, self-governing membership corporation. The Council has become the principal operating agency of both the National Academy of Sciences and the National Academy of Engineering in the conduct of their services to the government, the public, and the scientific and engineering communities. It is administered jointly by both Academies and the Institute of Medicine. The National Academy of Engineering and the Institute of Medicine were established in 1964 and 1970, respectively, under the charter of the National Academy of Sciences.

This study was prepared under EPA Contract #68-02-4073 and Department of Health and Human Services, Public Health Service Grant #ASU000001-06-31. The content of this publication does not necessarily reflect the views or policies of the U.S. Environmental Protection Agency or the Department of Health and Human Services, and an official endorsement should not be inferred.

INTERNATIONAL STANDARD BOOK NUMBER 0-300-03730-1

LIBRARY OF CONGRESS CATALOG CARD NUMBER 86-28622

Copyright © 1986 by the National Academy of Sciences

No part of this book may be reproduced by any mechanical, photographic, or electronic process, or in the form of a phonographic recording, nor may it be stored in a retrieval system, transmitted, or otherwise copied for public or private use, without written permission from the publisher, except for the purposes of official use by the United States Government.

Printed in the United States of America

202338021

## BOARD ON ENVIRONMENTAL STUDIES AND TOXICOLOGY

DONALD J. HORNING, Harvard University, Boston, Massachusetts,  
Chairman

ALVIN L. ALM, Thermal Analytical, Inc., Waltham,  
Massachusetts

RICHARD N. L. ANDREWS, University of North Carolina, Chapel  
Hill, North Carolina

WILLIAM E. COOPER, Michigan State University, East Lansing,  
Michigan

JOHN DOULL, University of Kansas Medical Center, Kansas City,  
Kansas

EMMANUEL FARBER, University of Toronto, Toronto, Ontario,  
Canada

BENJAMIN G. FERRIS, Harvard School of Public Health, Boston,  
Massachusetts

PHILIP LANDRIGAN, Mt. Sinai Medical Center, New York, New  
York

RAYMOND O. LOEHR, University of Texas, Austin, Texas

ROGER MINEAR, University of Illinois, Urbana, Illinois

PHILIP A. PALMER, E.I. DuPont de Nemours & Co., Wilmington,  
Delaware

EMIL PFITZER, Hoffman-La Roche, Inc., Nutley, New Jersey

PAUL PORTNEY, Resources for the Future, Washington, D.C.

PAUL RISSEK, Illinois Natural History Survey, Champaign, Illinois

WILLIAM H. RODGERS, University of Washington, Seattle,  
Washington

P. SHERWOOD ROWLAND, University of California, Irvine,  
California

LIANE B. RUSSELL, Oak Ridge National Laboratory, Oak Ridge,  
Tennessee

ELLEN SILBERGELD, Environmental Defense Fund, Washington,  
D.C.

PETER SPENOER, Albert Einstein College of Medicine, Bronx,  
New York

### National Research Council Staff

DEVRA LEE DAVIS, Acting Director, BEST

JACQUELINE PRINCE, Staff Associate

and cotinine in the plasma or urine of nonsmokers exposed to ETS are about 1 percent of the mean values observed in active smokers. Several studies have indicated that urinary cotinine concentrations in infants and children increase as the numbers of reported smokers increase in the home. At present, there may be difficulty in interpreting the relative cotinine levels in nonsmokers compared with smokers because of the reported slower clearance of cotinine in nonsmokers. Absorption, metabolism, and excretion of ETS constituents, including nicotine, need to be carefully studied in order to evaluate whether there are differences between smokers and nonsmokers in these factors. Further epidemiologic studies using biological markers are needed to quantify exposure-dose relationships in nonsmokers.

Thiocyanate, as measured in saliva, serum, or urine, does not appear to be sufficiently sensitive as an indicator of ETS exposure. Similarly, exhaled carbon monoxide and carboxyhemoglobin are not sufficiently sensitive to moderate or low levels of ETS exposure and thus are not particularly useful biological markers for exposure to ETS, except in experimental, acute exposure situations. There are several other sources of carbon monoxide in the environment that equal or exceed the concentrations of carbon monoxide attributable to ETS.

Other suggested biological markers of exposure are *N*-nitroso-proline, nitrosothioproline, and some of the aromatic amines that are present in high concentrations in SS. However, data on sensitivity and reliability of laboratory procedures for these markers are not sufficient to recommend their use at this time in epidemiologic studies of ETS.

Laboratory assays have shown mutagenic activity in the urine of smokers and ETS-exposed nonsmokers. The mutagenicity of urine is a function of many factors—such as dietary constituents, occupational exposures, and other environmental factors—which render any findings of mutagenicity nonspecific. Research is needed to clarify the appropriate methods for estimating mutagenicity and to isolate and identify the active agents in body fluids of ETS-exposed nonsmokers.

DNA adducts derived from tobacco-related chemicals can be measured in the blood. However, these chemicals, such as benzo[a]pyrene, are not unique to ETS. Studies are needed that can measure adducts of tobacco-specific chemicals.

2T2380202

## IN VIVO AND IN VITRO STUDIES

Laboratory studies can contribute to a better understanding of the factors and mechanisms involved in the induction of disease by environmental agents. There have been numerous bioassays conducted on MS. In examining the effects of MS, many research workers have used condensates of the smoke painted on the shaved skin of mice. This contrasts with the human exposure that is mainly in the respiratory tract. Nonetheless, these skin-painting studies have been useful in examining the carcinogenicity of different tobacco constituents and thus advancing knowledge of the actions of MS on a gross exposure level. Similar work with skin painting has not been done with ETS and would be of value for assessing the differential toxicity of ETS and MS.

In contrast to MS exposure, ETS exposure involves proportionately more exposure to gas phase than to particulate phase constituents. There have not, however, been studies of the effects of exposure to aged ETS. The relative *in vivo* toxicity of MS, SS, and ETS needs to be assessed.

Some studies have attempted to evaluate the gas phase of MS, SS, and ETS in short-term, *in vitro* assays. A solution of the gas phase of MS has been shown to induce dose-dependent increases in sister-chromatid exchanges in cultured human lymphocytes. Mutagenic activity has been found in the particulate matter of SS and in condensates of ETS. However, the work done to date is too sparse to permit any estimates of the mutagenicity of ETS per se, even though most of ETS consists of SS. Further *in vitro* assays of ETS are needed.

## HEALTH EFFECTS

This report reviews both chronic and acute health effects associated with ETS exposure in nonsmokers. Most epidemiologic studies of chronic health effects have been conducted on persons who have had long-term exposures to ETS from household members. The studies do not directly address chronic health effects in individuals who are exposed at work or have occasional exposures in the home or elsewhere.

Because the physicochemical nature of ETS, MS, and SS differ, the extrapolation of health effects from studies of MS or of

active smokers to nonsmokers exposed to ETS may not be appropriate.

Respiratory symptom studies in conjunction with epidemiologic investigations are needed to help clarify possible health effects of exposure to ETS in nonsmokers.

### Acute, Noxious Effects

The most common acute effects associated with exposure to ETS are eye, nose, and throat irritation, and objectionable smell of tobacco smoke. Tobacco smoke has a distinct and persistent odor, making control through ventilation particularly difficult. In closed rooms where smoking is allowed, a ventilation rate of greater than 60 cubic feet per minute per occupant is necessary to achieve air quality that is acceptable to more than 80% of adults entering the room as contrasted with rates of less than 10 cubic feet per minute per occupant when there is no smoking or other pollution. Annoyance with noxious tobacco odor largely governs the reactions of visitors, while occupants of smoky rooms are more likely to complain about irritating effects to the eye, nose, or throat. Particle filtration appears to lead to little or no decline in odor and irritation, suggesting that the effects are produced by gas-phase constituents. During exposure to ETS, eye blink rate is correlated with sensory irritation, such as burning eyes and nasal irritation. For some persons, eye tearing can be so intense as to be incapacitating. There is some evidence that nonsmokers are more sensitive to the noxious qualities of cigarette smoke than are smokers. Objective physiological or biochemical indices should be sought to validate reports of noxious reactions and chronic irritation associated with ETS.

Smoke contains immunogens, that is, substances that can activate the immune system. Approximately half of atopic (allergy prone) individuals react to various extracts of tobacco leaf or smoke presented in skin tests. However, the components of the extract that are responsible for this reaction have not been isolated. There is little correlation between positive reactions to skin tests and self-reported complaints of tobacco smoke sensitivity. Research is needed to evaluate the medical importance in atopic persons of these positive reactions to skin tests using ETS extracts and to relate immune response on skin tests to subjective complaints about the noxious, irritating properties of tobacco smoke.

### Respiratory Symptoms and Lung Function

Respiratory symptoms, such as wheezing, coughing, and sputum production, are increased in children of smoking parents. These symptoms are more common in children of smokers than children of nonsmokers. The largest studies place the increased risk of 20 to 80%, depending on the symptom being assessed and number of smokers in the household. Also, respiratory infections manifested as pneumonia and bronchitis are significantly increased in infants of smoking parents. Some studies have reported that infants of smoking parents are hospitalized for respiratory infections more frequently than children of nonsmokers. Among children aged under 1 year, studies are remarkably consistent in showing an increased risk of respiratory infections among children living in homes where parents smoke. There is a dose-response relationship that relates more to maternal smoking than paternal smoking. The association persists after allowing for possible confounding factors such as occupational data, respiratory illness in the parents, and birthweight. The mechanisms of the increased risk may either be a direct effect of ETS or due to a higher risk of cross-infection in such homes. Regardless of the mechanism, the exposure of small children to smoking in the home appears to put them at risk of respiratory illness.

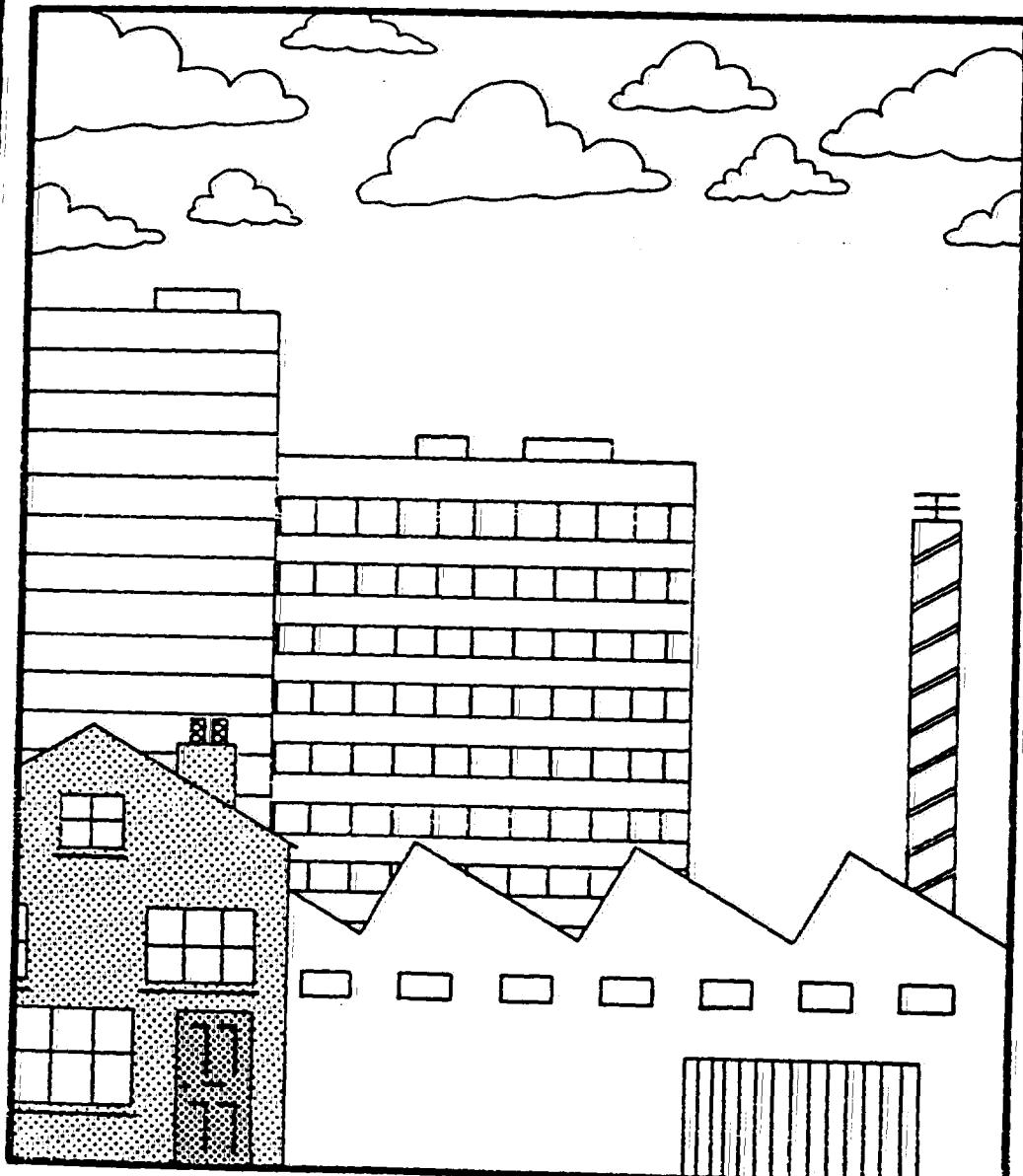
Since children exposed to ETS from parental smoking have an increased frequency of pulmonary symptoms and respiratory infections, it is prudent to eliminate ETS exposure from the environments of small children.

There is some evidence that parental smoking may affect the rate of lung growth in children. In children with one or more parents who smoke, lung function increase, which is a normal growth phenomenon, shows a small decrease in the rate of growth. An important issue currently unresolved is whether a child who is affected by exposure to ETS from parental smoking may be at an increased risk for the development of chronic airflow obstruction in adult life. In all studies of children, it is difficult to distinguish between the role of ETS exposure in utero and postnatally. Research is needed to address the issues of ETS exposure during childhood and fetal life and its possible relationship with airway hyperresponsiveness and pulmonary diseases in adult life.

B

2023380214

# INDOOR AND AMBIENT AIR QUALITY



2023380215

Edited by R. Perry  
and P.W. Kirk



# **INDOOR AND AMBIENT AIR QUALITY**

**Sold and distributed by Publications Division,  
Selper Ltd., 79 Rusthall Avenue,  
Chiswick, London W4 1BN**

**ISBN 0 948411 04 X**



**Copyright held by  
©Selper Ltd., 1988**

**Published by  
Publications Division, Selper Ltd., London  
Printed by Printext Ltd., London**

**2023380216**

# ENVIRONMENTAL TOBACCO SMOKE IN INDOOR AIR

P.W.W.Kirk, M.Hunter, S.O.Bock, J.N.Lester & R.Perry,  
Public Health Engineering, Imperial College, London SW7 2BU, U.K.

## ABSTRACT

A nationwide survey of ETS has been undertaken in the UK. Three components of ETS, namely particulate matter, nicotine and carbon monoxide were monitored in nearly 3,000 locations over 30 min periods in travel, work, home and leisure situations. Levels of the three components were generally low in comparison with UK Occupational Exposure Limits. In a follow-up study three methods of particulate measurement were compared in an intensive study of a few locations. It is concluded that MiniRAM particulate measurements with no impaction as used in the main survey are an over-estimate of true particulate levels.

## INTRODUCTION

There are many sources of indoor pollutants (both gaseous and particulate) including the use of gas stoves and fires, coal, coke, and wood fires, house plants, cooking, cleaning, painting, and the adoption of a variety of household and office products including cleaning agents, glues, correction fluids, plastics and varnishes (1, 2). In addition, the simple act of movement resuspends particulate matter (3) whilst building materials and furnishings, especially when new, may release a variety of organic materials into the indoor atmosphere (4). Release of formaldehyde from cavity wall insulation, furniture and fabrics are all examples of such indoor air pollutants and are of considerable public concern.

Of specific concern to some is Environmental Tobacco Smoke (ETS), which is a combination of sidestream smoke that is released from the cigarettes burning end and exhaled by smokers (5). The burning cigarette produces several thousand chemical compounds of which more than 3,800 have been identified (6). Cigarette smoke itself is an aerosol, consisting of a gaseous phase and a particulate phase with many of the chemical constituents being distributed between the two in a manner dependent upon their volatilities and solubilities (7). In the gaseous phase, the major constituents (by weight or volume) are nitrogen, oxygen and carbon dioxide, all normally present in the air. However, in the residual gases, apart from water vapour and the noble gases, carbon monoxide is present (approx. 4% by vol) together with small concentrations of other gases such as isoprene, acetaldehyde, acetone, hydrogen cyanide, toluene, acrolein and ammonia (8). In the particulate phase the major components are nicotine and tar, the latter being a complex mixture (7).

Particulate matter can be derived from sources other than tobacco products. A variety of combustion and condensation processes, amongst others, are associated

2023380212

2023380218

with particulate production. Indeed, these sources may originate from indoor or outdoor environments (9). Particulates may be classified according to their mode of formation as dust, smoke, fumes, fly ash, mist or spray (10). Environmental tobacco smoke particles are thought to have a median particulate mass of less than 1 µm in diameter (11, 12) which may vary as a consequence of different smoke age and environmental conditions (13). Particles of this size are not efficiently removed by such processes as sedimentation or impaction and hence remain in suspension, diffusion being the major removal mechanism although it is at its least efficient in the size range being considered here (8).

Three main techniques exist for the determination of particulate matter, namely gravimetric (filtration), piezoelectric balance (electrically charged particles are precipitated onto a vibrating quartz crystal changing its vibrational frequency in proportion to the deposited mass), and light scattering in the presence of particulates. The gravimetric technique is regarded as the reference technique but is unsuitable for use in widespread routine monitoring. Piezoelectric balance equipment is also impractical to use for large scale surveys due to its intrusive nature. The MiniRAM, relying on light scattering, is ideal for large surveys but is known to over-estimate particulate levels in ambient atmospheres containing ETS (14).

The aim of the study reported here was to examine the distribution of three representative components of ETS, namely particulate matter, carbon monoxide and nicotine, in a nationwide survey of smoking and non-smoking indoor atmospheres in the UK. A follow-up study compared the three principle techniques of determining particulate matter in selected environments together with the determination of the ETS marker, nicotine, to aid the interpretation of the nationwide survey.

#### MATERIALS AND METHODS

##### Study Design

For the 30 week field survey, the UK was divided into three major regions with roughly equivalent populations. Thirty sampling areas were derived from the most recent government statistics (15) by a market research group (MAS Survey Research Ltd., UK) with the number of sites in each region reflecting the population size. The areas sampled were selected to represent geographical, urban and social status within each region.

Monitoring was distributed between four situations, home (19%), work (25%), leisure (27%) and travel (29%). Routine air sampling procedures were performed by Hazleton Laboratories UK Ltd. (HUK) and were periodically verified by Imperial College staff using field sample cross checks. Each site was sampled over a 30 min period during each 10 week phase. The sample collection operatives were graduates employed by HUK and were rotated to a different region for each 10 week phase.

Homes were randomly pre-selected by MAS Survey Research Ltd., based on urban and social status after re-classification by local authority area, whereas work locations were chosen using a quota system. The quota system was determined by type and size (number of employees) of business reflecting the typical business at any location. Leisure and travel situations were identified and sampling of these was arranged around work and home samples. A balance of timing with respect to days of the week, start times, and times of the year was arranged to enable coverage of the spectrum of everyday environments. Smoking and non-smoking situations were sampled; the reported absence of smoking two hours prior to sampling determined a non-smoking location.

Subsequently, twelve follow-up sites were investigated, each for approximately 10 hrs duration in five specific types of environment (Table 1).

TABLE 1 : Details of sampled environments in the follow-up study

SITE NUMBER	SITE	TIME			SMOKING STATUS*	VENTILATION	No. OCCUPANTS
		START	FINISH	DURATION hrs			
1.	Office	08.30	18.30	10.0	NS	natural	1-4
2.	Office	08.30	18.30	10.0	NS	natural	1-4
3.	Office	09.00	19.00	10.0	S	natural	2
4.	Office	09.00	19.00	10.0	S	natural	2
5.	Bar (1)	11.15	20.45	9.5	S	natural	<75
6.	Bar (1)	12.30	22.00	10.0	S	natural	<75
7.	Bar (2)	12.15	22.00	9.75	S	natural/forced	<100
8.	Bar (2)	12.00	22.00	10.0	S	natural/forced	<100
9.	Flat	09.30	19.30	10.0	NS	natural	1
10.	Flat	08.30	18.30	10.0	NS	natural	1
11.	Workshop	09.30	21.30	12.0	NS	natural	1-5
12.	Workshop	08.30	20.30	12.0	NS	natural	1-5

\* NS - non-smoking      S - smoking

#### Equipment Selection and Analytical Procedures

The equipment was portable, robust and reliable, and capable of discreet operation to avoid abnormal behaviour patterns during the field sampling. A MiniRAM PDM-3 particulate monitor equipped with a flow through cell (CCA Corp., USA) with no impactor, was interfaced to a Squirrel data logger (Grant Inst. Ltd., UK), storing the particulate data every 2 mins for the duration of each 30 min sampling period. Air flow was maintained by an Alpha pump (DuPont Ltd., USA) operating at  $0.015 \text{ m}^3 \text{ h}^{-1}$ . Concomitantly, a second Alpha pump drew air at  $0.006 \text{ m}^3 \text{ h}^{-1}$  via a Perkin Elmer ATD50 stainless steel sampling tube containing 200 mg of Tenax TA (Chrompack Ltd., UK) for the collection of nicotine and then through a carbon monoxide dosimeter (General Electric 15, ECOSICO<sub>2</sub>; MDA Scientific, UK), the latter also being connected to the Squirrel logger.

During the follow-up monitoring detailed particulate matter data was obtained over longer periods using three different techniques. Gravimetric data was obtained by drawing air at  $28.1 \text{ min}^{-1}$  through a pair of 47 mm, 0.5  $\mu\text{m}$  pore size

2023380219

2023380220

PTFE membrane filters (Amicon Ltd., UK) connected in parallel using a double headed diaphragm pump (Charles Austin Ltd., UK) fitted in a sound proof ventilated wooden enclosure. Filters were preconditioned by drying in a dessicator to constant weight. Following sampling, filters were similarly dried prior to reweighing. Simultaneously, particulate matter was determined using three MiniRAM monitors, two of which were fitted with particle impactors (TSI Inc., UK) to remove particles with nominal diameters of 3.5 µm and 1.0 µm respectively when operated at 2.0 l min<sup>-1</sup> (11), the third having no impactor. Each MiniRAM was zeroed prior to each sampling period using a 0.5 µm membrane filter inline, checked and rezeroed periodically as required. To determine the relative response of each MiniRAM, the three were connected in parallel with no impactors in a number of laboratory experiments in a controlled environment. MiniRAM results are corrected to give equivalent response.

An additional particulate monitor, a Model 3500 Piezobalance Respirable Aerosol Mass Monitor (TSI Inc., UK) was operated using a 2 min integrated monitoring time to coincide with MiniRAM readings (12 readings for each MiniRAM taken using 10 sec integration times). An impactor with a 3.5 µm cut-off was an integral part of the piezobalance. The piezoelectric crystal in the monitor was cleaned with detergent and distilled water in accordance with the manufacturers instructions prior to each reading. Cleaning with dilute ammonia was found to be necessary after sampling in some locations to maintain the frequency of oscillation near to the baseline frequency. MiniRAM and piezobalance readings as described above were taken every 15 min for the duration of the follow-up monitoring periods.

Nicotine samples taken during the follow-up study were obtained over hourly periods and composite samples were collected over each complete sampling period. Nicotine collected on the ATD 50 Tenax sampling tubes were analysed using 2-stage thermal desorption using a Perkin Elmer ATD50 linked to a Perkin Elmer 8320 capillary column GC with a flame ionization detection (16). An air volume of 15 l was sampled for the hourly and period samples in the follow-up study giving a detection limit of 0.8 µg m<sup>-3</sup> compared with a detection limit of 13.6 µg m<sup>-3</sup> in the nationwide field survey.

Statistical analysis of the main field study was undertaken on the Imperial College mainframe computer. For the follow-up study an Apple Macintosh SE was used running Statworks TM software.

#### RESULTS AND DISCUSSION

Laboratory studies of ETS using simulated 'real-life' conditions are intended to overcome the inherent problems associated with sampling uncontrolled environments in field sampling. However, experience has shown that such controlled experiments cannot substitute for ETS data acquired directly in the field. No previous study has attempted this on a large scale; most have considered relatively small numbers of samples in a small number of environments.

Summary data for the complete 30 week field study is included in Table 2, which presents indoor air exposures under a variety of activity modes. In the table a smoking sample is one in which smoking is known to have occurred during sampling or within the two hours prior to sampling. Collected data from the field study were grouped by site and location. Although approximately 50% of the 2912 samples were classified as smoking, imbalances between smoking and non-smoking situations were evident. For example, 39% of office samples were classified as smoking compared with 90% of those taken in restaurants. Overall, higher

TABLE 2 : Summary for 30 week study

ACTIVITY	TEMP C	REL. %	CO(ppm)			TPM(mg/m <sup>3</sup> )*			NICOTINE(ug/m <sup>3</sup> )**		
			SM(T)	SM(Y)	SM(N)	SM(T)	SM(Y)	SM(N)	SM(T)	SM(Y)	SM(N)
TRAVEL	MEAN	20	44	2.8	2.9	2.7	.62	.79	.42	17	24
	SD	3	8	2.8	2.5	3.1	.64	.75	.36	31	40
	MIN	1	22	.0	.0	.0	.00	.00	.07	7	7
	MAX	34	75	17.4	13.1	17.4	4.98	4.98	1.83	416	414
	DATA	343	308	518	283	235	538	297	241	364	313
WORK	MEAN	20	45	2.1	2.2	2.1	.41	.61	.31	10	14
	SD	4	9	2.7	3.3	2.4	.42	.59	.26	12	18
	MIN	8	23	.0	.0	.0	.00	.07	.00	7	7
	MAX	30	75	31.9	31.9	21.9	5.78	5.78	2.20	167	167
	DATA	723	721	671	221	450	704	224	480	733	238
HOME	MEAN	20	46	1.9	2.3	1.8	.36	.70	.27	10	19
	SD	3	9	2.3	2.9	2.1	.36	.52	.23	17	33
	MIN	10	5	.0	.0	.0	.00	.07	.00	7	7
	MAX	30	71	26.2	26.2	25.4	3.15	3.15	2.05	292	292
	DATA	766	763	688	139	549	748	156	592	774	612
LEISURE	MEAN	20	45	2.7	2.8	2.2	.84	.91	.33	20	22
	SD	3	8	2.7	2.7	2.6	.82	.85	.26	29	31
	MIN	8	17	.0	.0	.0	.07	.07	.07	7	7
	MAX	32	75	28.7	28.7	18.9	6.22	6.22	1.24	450	450
	DATA	819	578	780	676	104	811	703	108	841	729
TOTAL	MEAN	20	45	2.4	2.7	2.1	.56	.81	.31	16	21
	SD	4	9	2.7	2.8	2.5	.63	.77	.27	24	32
	MIN	8	5	.0	.0	.0	.00	.00	.00	7	7
	MAX	34	75	31.9	31.9	25.4	6.22	6.22	2.20	450	450
	DATA	2853	2370	2657	1319	1338	2801	1380	1421	2912	1642
											1470

NOTE \*: Particulate matter measured by MinIRAM

\*\*: Nicotine data below detection limit included as 6.8 ug.m<sup>-3</sup>

## Percentiles for 30 week study

PERCENTILES	CO(ppm)			TPM(mg/m <sup>3</sup> )			NICOTINE(ug/m <sup>3</sup> )*		
	SM(T)	SM(Y)	SM(N)	SM(T)	SM(Y)	SM(N)	SM(T)	SM(Y)	SM(N)
MINIMUM	.0	.0	.0	.00	.00	.00	N.D.	N.D.	N.D.
01% VALUE	.0	.0	.0	.07	.07	.07	N.D.	N.D.	N.D.
05% VALUE	.0	.0	.0	.07	.15	.07	N.D.	N.D.	N.D.
10% VALUE	.0	.0	.0	.15	.22	.15	N.D.	N.D.	N.D.
25% VALUE	.5	.8	.3	.22	.37	.15	N.D.	N.D.	N.D.
50% VALUE	1.9	2.1	1.6	.37	.59	.22	N.D.	N.D.	N.D.
75% VALUE	3.4	3.8	3.1	.66	1.02	.37	N.D.	23.3	N.D.
80% VALUE	3.9	4.2	3.5	.81	1.17	.39	15.2	28.4	N.D.
90% VALUE	5.1	5.6	4.5	1.24	1.65	.59	30.7	46.5	N.D.
95% VALUE	6.8	7.2	5.9	1.68	2.42	.88	49.8	76.4	16.4
99% VALUE	11.5	12.6	11.5	3.07	3.81	1.46	112.4	146.0	36.1
MAXIMUM	31.9	31.9	25.4	6.22	6.22	2.20	449.9	449.9	98.5
DATA	2657	1319	1338	2801	1380	1421	2912	1642	1470

NOTE \*: Particulate matter measured by MinIRAM

\*\*: 77.5% of the nicotine data are below detection limit

- SD: Standard deviation  
 MEAN: Arithmetic mean  
 DATA: Number of observations  
 SM(T): Combined smoking and non-smoking situations  
 SM(Y): Smoking situations only  
 SM(N): Non-smoking situations only

2023380221

2023380222

incidence of smoking occurred in travel and leisure situations, the converse being true for work and homes.

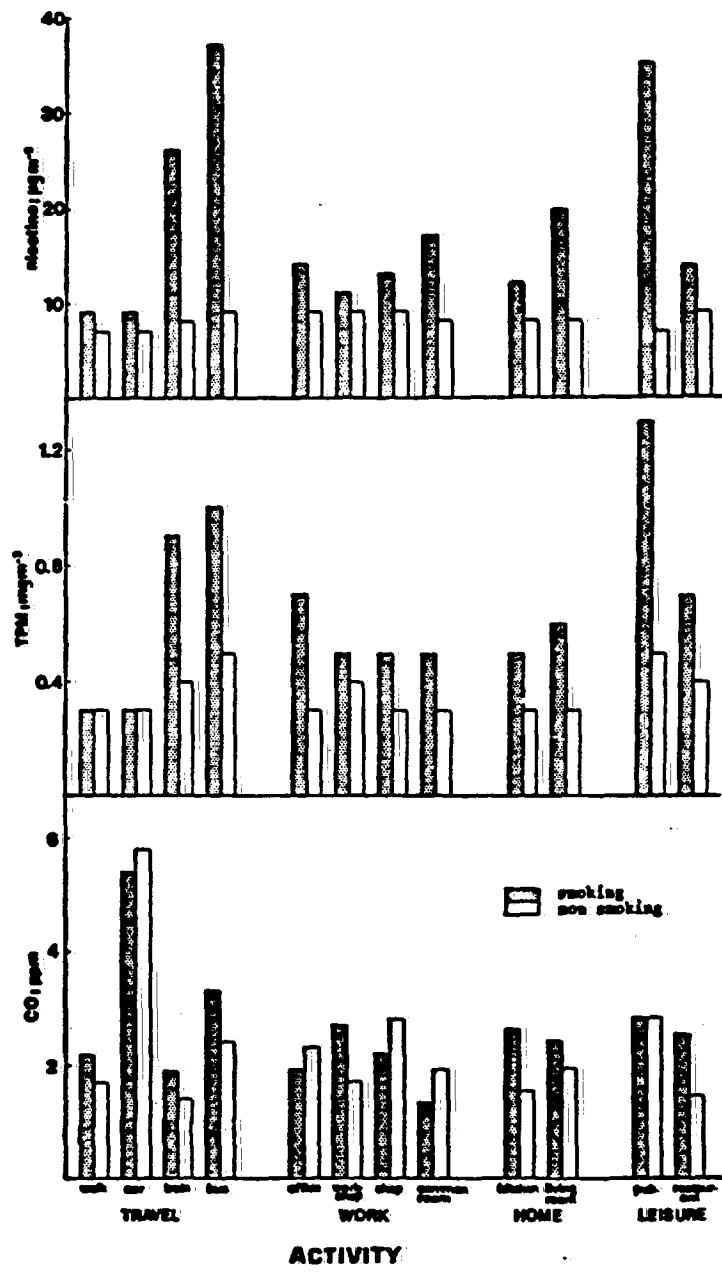
During the field study, for each classification of travel, work, home and leisure, with the exception of travel by car and walking, MiniRAM measurements were observed to be greater in smoking situations than non-smoking ones (Figure 1). Differences between smoking and non-smoking MiniRAM total particulate matter values were observed in pubs, trains, buses, offices and restaurants (Figure 1). Overall MiniRAM values in non-smoking situations were  $0.3 \text{ mg m}^{-3}$  except for travel which was  $0.4 \text{ mg m}^{-3}$ . Where smoking occurred the mean MiniRAM particulate level was between  $0.6$  and  $0.9 \text{ mg m}^{-3}$  the highest levels being associated with leisure situations.

Over-estimates for MiniRAM particulate data have been reported in the literature (14). When considering the particulate levels as measured by the MiniRAM, the relationship between the various techniques of measuring particulate matter must be considered. Since the light scattering effect is dependent upon particulate size and reflectivity it is apparent that the results obtained using the MiniRAM in the main survey are subject to a correction factor the magnitude of which is likely to be affected by the particular environment being sampled. A summary of the results obtained using the various methods of particulate monitoring in specific environments is included in Table 3. An examination of 15 min values applying the Kolmogorov-Smirnov test demonstrated that the individual sampling data sets were not normally distributed, although an examination of all the data for a particular set of conditions (e.g. piezobalance data for Sites 1 to 12) showed these data sets to be one tail of the normal distribution 'bell shape'. Therefore the Wilcoxon Signed Rank test was applied to the paired non-parametric data sets to compare the various MiniRAMS with the piezobalance response in the 12 tests. Nearly all the responses were highly significantly different ( $P=0.01$  or lower) with a few exceptions in the non-smoking low particulate level sites.

It is apparent from Table 3 that the ratio of response when comparing the MiniRAM (with no impactor) as used in the field study, and the gravimetric data varies greatly in the different environments. The highest ratio of 4.8 was found in the bar while the lowest, 0.36 was observed in the workshop. It is evident that the nature of the particulate matter in these two environments is quite dissimilar. The mean ratio for all twelve locations was 2.0 with a standard deviation of 1.2. In contrast a comparison of the piezobalance and gravimetric data gave an overall ratio of response of 0.82 with a standard deviation of 0.38 (Table 3). Considering the intermittent nature of the piezobalance (and MiniRAM) data in comparison with the integrated nature of the gravimetric data an identical 1:1 relationship would not, however, be expected.

Therefore, on the basis of the follow-up study, it may be concluded that the MiniRAM with no impactor, as used in the nationwide field survey, over-estimated the particulate matter concentration by on average a factor of 2.0 compared with gravimetric data and 2.5 compared with piezobalance measurements. Taking the factor of 2.0 into account, the corrected mean particulate levels in each of the smoking locations in the nationwide survey, may be compared with the corrected non-smoking situation (shown in brackets); travel  $0.40$  ( $0.21$ )  $\text{mg m}^{-3}$ , work  $0.30$  ( $0.16$ )  $\text{mg m}^{-3}$ , home  $0.35$  ( $0.14$ )  $\text{mg m}^{-3}$  and leisure  $0.46$  ( $0.17$ )  $\text{mg m}^{-3}$ . These corrected results are comparable with previous findings. Repace and Lourey (17) reported mean particulate levels between  $0.09$  and  $0.11 \text{ mg m}^{-3}$  in four restaurants and between  $0.09$  and  $0.70 \text{ mg m}^{-3}$  in public buildings, while Weber and Fischer (18) found a mean particulate level of  $0.13 \text{ mg m}^{-3}$  in 44 offices with a range from  $0.01$  to  $1.13 \text{ mg m}^{-3}$ . Sterling et al., (9), however, reported a somewhat lower mean particulate level of  $0.038 \text{ mg m}^{-3}$  ( $n=81$ , range =

**Figure 1 : Mean CO, MiniRAM TPM and nicotine values by situation and location**



2023380223

TABLE 3 : Summary of results for sampled environments in the follow-up study

SITE	SMOKING	MINIRAM PARTICULATE MATTER ( $\text{mg m}^{-3}$ )			PIEZOBALANCE ( $\text{mg m}^{-3}$ )	GRAVIMETRIC ( $\text{mg m}^{-3}$ )	NICOTINE ( $\mu\text{g m}^{-3}$ )	
		NO IMPINGER	3.5 $\mu\text{m}$ CUT-OFF	1.0 $\mu\text{m}$ CUT-OFF			HOURLY SAMPLES	PERIOD SAMPLE
1. Office	NS	0.12*	0.05 (0.03)	0.09 (0.06)	0.06 (0.02)	0.077	1.0* (0.1)**	1.2
2. Office	NS	0.05 (0.03)	0.06 (0.06)	0.02 (0.03)	0.05 (0.01)	0.046	1.2 (0.3)	0.8
3. Office	S	0.26 (0.27)	0.28 (0.30)	0.23 (0.26)	0.11 (0.06)	0.120	9.7 (16)	5.7
4. Office	S	0.26 (0.20)	0.26 (0.21)	0.25 (0.19)	0.10 (0.05)	0.117	2.2 (2.3)	3.3
5. Bar (1)	S	0.14 (0.17)	0.11 (0.15)	0.06 (0.10)	0.07 (0.05)	0.109	4.7 (4.2)	4.0
6. Bar (1)	S	0.30 (0.24)	0.35 (0.25)	0.27 (0.24)	0.07 (0.04)	0.130	8.5 (4.7)	6.8
7. Bar (2)	S	0.40 (0.46)	0.41 (0.51)	0.35 (0.43)	0.15 (0.11)	0.083	7.2 (5.6)	5.1
8. Bar (2)	S	0.32 (0.43)	0.39 (0.54)	0.34 (0.49)	0.08 (0.08)	0.155	13 (17)	12.0
9. Flat	NS	0.20 (0.05)	0.27 (0.06)	0.22 (0.09)	0.05 (0.02)	0.069*	6.3 (1.9)	<0.8
10. Flat	NS	0.15 (0.43)	0.18 (0.51)	0.12 (0.39)	0.06 (0.12)	0.069*	5.7(-)	5.6
11. Workshop	NS	0.04 (0.03)	0.04 (0.03)	0.01 (0.01)	0.05 (0.02)	0.110	2.3 (0.7)	1.8
12. Workshop	NS	0.16 (0.33)	0.10 (0.29)	0.05 (0.20)	0.08 (0.07)	0.140	1.8 (0.7)	1.5

NS = non-smoking; S = smoking; \* = mean; \*\* = standard deviation; + = data obtained over both periods.

2023380224

MD-0.7 mg m<sup>-3</sup>) in buildings where smoking was permitted and a mean of 0.038 mg m<sup>-3</sup> (n=20, range = 0.014-0.32 mg m<sup>-3</sup>) where smoking was restricted.

All particulate concentrations in the nationwide survey were below the 'total' dust Occupational Exposure Limit (OEL) of 10 mg m<sup>-3</sup> (set for an 8 hour period), and assuming a correction factor of 2.0 on average, all were also below the OEL of 5 mg m<sup>-3</sup> set for 'respirable dust' set by the UK Health and Safety Executive (19). Furthermore, 95% of all survey samples were less than 9% of the 'total' dust OEL and 95% of all smoking samples would be less than 12% of the OEL. Statistically higher levels of MiniRAM particulate matter were observed in the smoking environment compared with the non-smoking, for each of the four activity types.

The observed behaviour of occupants in the office environment in the follow-up study enables a direct comparison between smoking and non-smoking to be made (Figure 2). When smoking was not occurring, all the methods of particulate measurement found levels below 0.22 mg m<sup>-3</sup> with 15 min fluctuations remaining relatively small during both experimental periods. In contrast, when smoking occurred, marked fluctuations were observed during the sampling period. This was particularly evident for the MiniRAM data regardless of the presence of absence of impactors (Figure 2). Particulate levels in the bars exhibited significant peaks associated with the hours of opening and hence the times of ETS exposure and the preparation of cooked snacks (Figure 3), while operation of a specific milling machine in the workshop produced comparable levels of particulates (Figure 3).

Correlation coefficients between the 15 min MiniRAM values and piezobalance results in the follow-up study were generally highly significant ( $P=0.01$  or lower) with the exception of the non-smoking office locations (Site 1 and 2) and the non-smoking flat (Site 9). In particular in the case of Sites 1 and 9 no significant correlation was evident between the piezobalance results and the MiniRAMS with 3.5  $\mu\text{m}$  and 1.0  $\mu\text{m}$  impactors. It should be noted that the MiniRAMS had a readout resolution of 0.02 mg m<sup>-3</sup> on the digital display and a precision and stability of  $\pm 0.02$  mg m<sup>-3</sup> over the 2 min sampling periods. The gravimetric particulate determinations obtained in locations 1,2 and 9 were the lowest observed in the follow-up study. The effects of such discrete data in combination with instrumental variation would be expected to be most significant at low particulate levels.

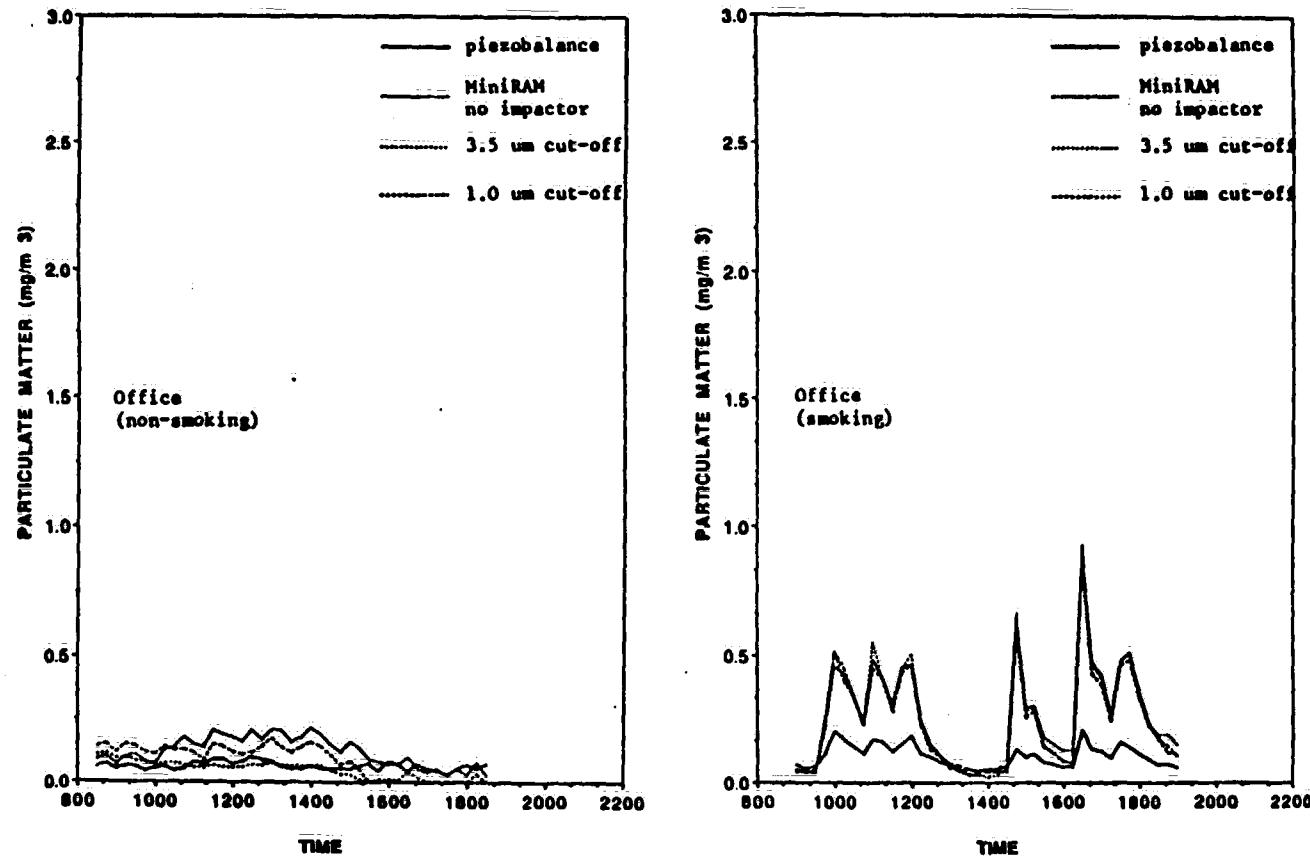
More directly comparable in terms of assessing differences between the methods of determining particulate levels is the MiniRAM with a 3.5  $\mu\text{m}$  impactor and the piezobalance which has a 3.5  $\mu\text{m}$  impactor as an integral component. The relative response when comparing these two methods in the follow-up study was on average 2.65 with a standard deviation of 1.59. The highest ratios were observed in the bars and the lowest in the non-smoking workshop and flat. Since the particulate size of ETS is generally thought to be less than 1.0  $\mu\text{m}$ , the MiniRAM with a cut-off at 1.0  $\mu\text{m}$  would be expected to indicate the presence of smoking when for example, it is expressed at a proportion of the MiniRAM response with a cut-off at 3.5  $\mu\text{m}$ . Indeed the lowest ratios of 1.0 : 3.5  $\mu\text{m}$  MiniRAMS were observed in non-smoking locations and in general the highest ratios were in smoking locations, although the highest ratio which was inexplicably greater than unity was observed in the non-smoking office (Site 1 on Table 3). The mean response of the 1.0  $\mu\text{m}$  cut-off MiniRAM relative to the 3.5  $\mu\text{m}$  cut-off MiniRAM was 0.77 with a standard deviation of 0.40, the lowest ratio being 0.25 for the non-smoking workshop.

In calculating nicotine values for the nationwide field survey a value of 6.8  $\mu\text{g m}^{-3}$  (i.e. half the limit of detection) was assumed whenever the nicotine

2023380225

2023380226

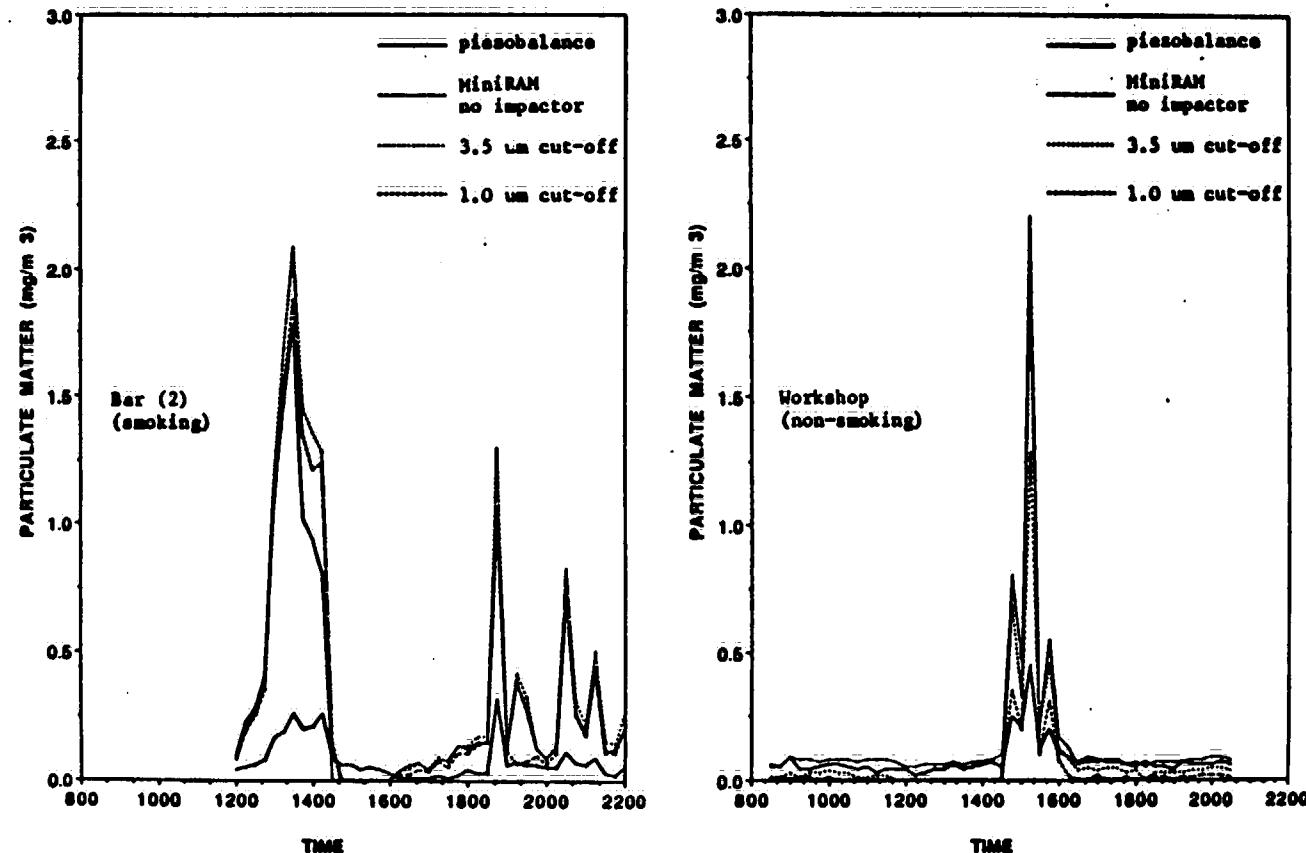
Figure 2. Particulate measurements by piezobalance and MiniRAM during the follow-up study



108

2023380227

Figure 3. Particulate measurements by piezobalance and MiniRAMS during the follow-up study



109

level was below the limit of detection. It is likely that this led to an over-estimation of nicotine levels as more than 77% of the nicotine samples were below detectable levels. Indeed, all of the period mean nicotine concentrations in the follow-up study would have been below the detection limit of the main survey (Table 2). Furthermore, the measurement of nicotine concentrations in the flat in the order of  $6 \text{ }\mu\text{g m}^{-3}$  suggests the presence of interfering volatile organics as no smoking was known to have occurred in the flat for several months prior to sampling. The flat was of recent construction and the interference was assumed to be volatile emissions from building materials such as paint (20). In the nationwide survey a similar pattern emerged to that observed for particulate matter, with higher levels associated with travel and leisure were smoking occurred. The highest mean nicotine concentrations were observed in trains, busses and public houses classified as smoking locations (Figure 1). Overall, mean nicotine concentrations of 24, 22, 19 and  $14 \text{ }\mu\text{g m}^{-3}$  were found for travel, leisure, home and work smoking locations respectively compared with between 7 and  $9 \text{ }\mu\text{g m}^{-3}$  in non-smoking locations.

No individual nicotine concentration exceeded the OEL set for occupational exposure to nicotine of  $500 \text{ }\mu\text{g m}^{-3}$ , and 95% of all locations were below 10% of the 8-hour time weighted average OEL (19). Previous studies using field sampling have produced comparable nicotine concentrations to those reported here. Weber and Fischer (18) reported a mean nicotine concentration of  $1.1 \text{ }\mu\text{g m}^{-3}$  in various workplaces (maximum  $16 \text{ }\mu\text{g m}^{-3}$ , n=160) while Muramatsu et al. (21) reported a mean nicotine concentration of  $20.3 \text{ }\mu\text{g m}^{-3}$  (maximum  $83 \text{ }\mu\text{g m}^{-3}$ , n=91) in work, leisure and travel situations. More recently, Hammond et al. (22) found a mean nicotine concentration of  $5.1 \text{ }\mu\text{g m}^{-3}$  (maximum  $41 \text{ }\mu\text{g m}^{-3}$ , n=14) in railway workshops and  $19 \text{ }\mu\text{g m}^{-3}$  (maximum  $48 \text{ }\mu\text{g m}^{-3}$ , n=10) in offices. In another survey of office environments, Sterling et al. (9) reported a median nicotine concentration of  $8.5 \text{ }\mu\text{g m}^{-3}$  (maximum  $53 \text{ }\mu\text{g m}^{-3}$ , n=10).

Mean carbon monoxide levels were not significantly different in smoking and non-smoking environments in the nationwide field survey (significance level of 0.01, corresponding to an overall significance of about 0.05). The highest mean carbon monoxide values of 5 to 6 ppm, observed during car journeys, were independent of smoking. In non-smoking environments, carbon monoxide levels associated with travel were statistically significantly higher than for work or home. No such differences were observed in smoking situations.

Similar levels of carbon monoxide to those found in this study have been reported elsewhere (23). Mean carbon monoxide levels in both smoking and non-smoking offices have been recorded at 2.5 ppm, while carbon monoxide levels up to 21 ppm in bus garages, 89 ppm in car ferries and 68 ppm in warehouses have been noted (23). These data and others suggest that carbon monoxide levels can be appreciably affected by sources other than ETS. In the nationwide study reported here, 95% of all carbon monoxide concentrations were less than 15% of the 8 hour OEL set at 50 ppm in the UK (19) and none exceeded it.

#### CONCLUSIONS

An extensive 30 week field survey has been carried out of the levels of particulate matter, nicotine and carbon monoxide in indoor environments throughout the UK. Home, travel, work and leisure environments have been examined, with an approximately even distribution of measurements between smoking and non-smoking locations. When the results were classified according to activity, it was found that leisure and travel tended to be associated with higher levels than home or work. Overall no significant difference was found between smoking and non-smoking locations for carbon monoxide in each of the

2023380228

four types of situations, but MiniRAM particulate and nicotine levels were higher in smoking situations. Results for non-smoking environments showed that there are sources of particulate matter and carbon monoxide other than ETS. As reported the amounts of the three components in the field survey were generally low in comparison with UK OELs and were often close to detection limits of the equipment used. Taking the adjustment from the follow-up study into account, particulate levels were consistent with previous studies reported in the literature.

A comparison of three methods for the evaluation of particulate matter, namely MiniRAM particulate dosimeters, piezobalance aerosol monitors and a gravimetric procedure in an intensive study of a small number of environments demonstrated that MiniRAM particulate monitors over-estimate the contribution of ETS to indoor particulate levels. This is likely to be related to the high reflectance of ETS particulates. Nicotine concentrations determined in follow-up studies with an improved detection limit, were in general lower than those observed in the nationwide field survey. This suggests that the nicotine levels reported in the field survey were somewhat over-estimated, particularly for the non-smoking locations.

#### REFERENCES

1. T.D. Sterling and D. Kobayashi, Environ. Res., 13, 1-35 (1977).
2. National Academy of Sciences/National Research Council, Indoor Pollutants, National Academy Press, Washington DC, USA (1981).
3. J.R. Girman, A.T. Hodgson and M.L. Ward, Atmos. Environ., 21, 315-320 (1987).
4. L.A. Wallace, E. Pellizzari, B. Leaderer, H. Zelon and L. Sheldon, Atmos. Environ., 21, 385-394 (1987).
5. M.W. First, Constituents of sidestream and mainstream tobacco smoke and markers to quantify exposure to them in Indoor Air and Human Health, R.B. Gamble and S.V. Kaye, eds., Chelsea M.I. Lewis Publishers, 195-203 (1985).
6. National Research Council, Environmental Tobacco Smoke, Measuring Exposures and Assessing Health Effects, National Academy Press, Washington DC, USA (1986).
7. M.R. Guerin, C.E. Higgins and R.A. Jenkins, Atmos. Environ., 21, 291-297 (1987).
8. US Surgeon General, The Health Consequences of Involuntary Smoking, US Dept. of Health and Human Services, US Govt. Printing Office, Washington DC, USA (1986).
9. T.D. Sterling, C.W. Collett and E.M. Sterling, J. Occupat. Med., 29, 57-62 (1987).
10. R.M. Harrison, Analysis of Particulate Pollutants in Handbook of Air Pollution Analysis, second edition, R.M. Harrison and R. Perry, eds., Chapman and Hall, London, 155-203 (1986).
11. M.F. Dube and C.R. Green, Recent Adv. Tob. Sci., 8, 42-102 (1982).
12. Y. Ishizu, K. Kaneki and T. Okadi, J. Aerosol Sci., 18, 123-129 (1987).
13. W.C. Binds, Am. Ind. Hyg. Assoc. J., 39, 48-54 (1978).
14. R.G. Rawbone, W. Burns and R.A. Patrick, Toxicol Letts., 35, 125-129 (1985).
15. Office of Population Censuses and Surveys (Social Survey Division), General Household Survey 1983, HMSO, London, UK (1985).
16. R.E. Bell, Intern. J. Environ. Anal. Chem., (in press), (1988).
17. J.C. Repace and A.H. Lowrey, Science, 208, 464-472 (1980).
18. A. Weber and T. Fischer, Int. Arch. Occup. Environ. Health, 47, 209 (1980).
19. Health and Safety Executive, Occupational Exposure Limits, Guidance Note EH40/87, HMSO, London, UK (1987).

2023380229  
628080

20. M.K. Hansen, M. Larsen and K.H. Cohn, Scand. J. Work Environ. Health, 13, 473-485 (1987).  
21. M. Muramatsu, S. Umemura, T. Akata and H. Tomita, Environ. Res., 35, 218-227 (1984).  
22. S.K. Hammond, B.P. Leaderer, A.C. Roche and M. Schenker, Atmos. Environ., 21, 457-462 (1987).  
23. T.D. Sterling, H. Dimich and D. Kobayashi, J. Air Pollut. Control Assoc., 32, 250-259 (1982).

2023380230

by experience, patients who by a combination of antipsychotic drugs in skilled institutions or psychiatric hospitals. probably the experience of the other psychiatrists who, like me, continue to use combined therapy in accordance with the guidelines to the con-

cerned authorities in the field either explain why these combinations are not frequent and/or do not present rational use or change advice in their future editions. There is seldom indication to the use of antipsychotics with the side effect profile, but there are frequent indications to combine with different profiles," and these are the indications. Only then will government reviewers change current "indicator" of inappropriate drug prescribing and will clinicians be relieved of their current concern of being faulted by reviewers for rational prescribing practice.

Lewis Glickman, MD  
Bronx, NY

Well CP, Glass RM: Concomitant antipsychotic drugs. *JAMA* 1983;250:1232.  
Judd RL, Jackson AH: Approaches to schizophrenia. In: Judd RL, Jackson AH (eds): *Approaches to Schizophrenia*. Baltimore, Williams & Wilkins Co, 1980, pp 2261-2262.  
2. Jeste DV, Wyatt RJ: *Understanding and Treating tardive dyskinesia*. New York, Guilford Press, 1982, pp 289-300.

Given the present state of knowledge about this issue, we believe that the concomitant use of two different antipsychotic drugs should be a matter for the clinical judgment of the prescribing physician. Most important are the identification of indications for the use of antipsychotic drugs, monitoring for adverse effects, and careful assessment of the need to continue treatment with these drugs for more than a few months.<sup>1</sup>

RICHARD M. GLASS, MD  
CHARLES P. KORNBLITH, MD  
University of Chicago  
Pritzker School of Medicine

1. Davis JM: Antipsychotic drugs, in Kaplan HL, Freedman AM, Sadock BJ (eds): *Comprehensive Textbook of Psychiatry*, ed 2. Baltimore, Williams & Wilkins Co, 1980, pp 2261-2262.
2. Jeste DV, Wyatt RJ: *Understanding and Treating tardive dyskinesia*. New York, Guilford Press, 1982, pp 289-300.

### Costs and Benefits of Cesarean Sections

To the Editor.—Dr Sachs et al<sup>1</sup> made a nice contribution with their article on cesarean section. The cost-morbidity angle of studies on the delivery of low-birth weight and breech infants needs to encompass those infants who do not die but have permanent neurological damage and live a very long time at a tremendous expense.

G. C. TOW NABORS, MD  
Dallas

1. Sachs BP, McCarthy BJ, Rubin G, et al: Cesarean section: Risk and benefits for mother and fetus. *JAMA* 1983;250:2157-2159.

In Reply.—Dr Nabors' kind words are appreciated. We were unable to assess either the cost of long-term care for infants that do not die or the decreased cost for those infants less traumatized because of a cesarean delivery. We therefore emphasized that our study was not meant to be a cost-benefit analysis but an opening shot in a discussion that needs to take place.

BENJAMIN P. SACHS, MD, BS, DFACD  
Harvard Medical School  
Beth Israel Hospital  
Boston

### Passive Smoking and Uptake of Carbon Monoxide in Flight Attendants

To the Editor.—There is concern about the health effects of passive smoking.<sup>1</sup> Because of concern among flight attendants about passive exposure to cigarette smoke during work on commercial aircraft, a preliminary investigation was conducted to search for an increase in expired air (end tidal) carbon monoxide in flight attendants after work. Expired air (end tidal) carbon monoxide was chosen

because carbon monoxide concentration is known to vary linearly with the rate of cigarette burning in an environment.<sup>2</sup>

Volunteers gave their informed consent after the nature of the procedures had been explained to them. Nonsmoking volunteer flight attendants filled out health history questionnaires before flight and recorded their observations during flight. Expired air (end tidal) carbon monoxide was measured before and after each flight for each volunteer. All flights were "turnaround" flights from Los Angeles to Honolulu and back. These flights were of about five hours' duration in each direction, with an hour on the ground in Honolulu.

The volunteers were 16 women between 25 and 48 years of age. Four of them worked in nonsmoking sections only during the flights of interest. There was no increase in the concentration of carbon monoxide in the expired air (end tidal) of these flight attendants during the flights in this study. In fact, their exhaled air carbon monoxide levels decreased by an insignificant amount. When the four attendants who worked in the nonsmoking areas were excluded from the analysis, the results were not altered substantially. These results are consistent with results of a similar study reported in 1983<sup>3</sup> and an earlier study involving a larger number of subjects.<sup>4</sup> These results indicate that the concentration of smoke to which flight attendants are passively exposed is too low to alter significantly their expired air carbon monoxide levels. Other possible health effects of such exposure were not addressed.

DOUGLAS B. DUNCAN, MD  
Peter P. O'BRIEN, MD  
University of California, Irvine  
Orange Occupational Health Center

1. White JR, Freed HF: Small airway dysfunction in non-smokers chronically exposed to tobacco smoke. *N Engl J Med* 1980;302:720-723.
2. Hedges L: Cigarette smoke in closed spaces. *Environ Health Perspect* 1972;2:117.
3. Fuleihan GH, Benowitz NL, Becker CE: Passive absorption of nicotine in airline flight attendants. *N Engl J Med* 1983;308:1105.
4. Report on Health Aspects of Smoking in Transport Aircraft. Dept of Health and Human Services, December 1981.

### Foreign Body in a Meckel's Diverticulum

To the Editor.—A 19-month-old girl infant was seen because of abdominal pain and discomfort. Her mother stated that approximately six weeks ago she had swallowed a penny. Roentgenograms revealed the coin in the right lower quadrant. The patient continued to have intermittent bouts of abdominal pain and vomiting. Seri-

al roentgenograms showed no progress. Exploration was undertaken and the coin was found in a diverticulum. The diverticulum, with the coin, was excised. Recovery was uneventful. A wound abscess developed and required drainage.

### Access to Medical Care

To the Editor.—In his letter "A New Physician Shortage," Dr Ginzberg<sup>1</sup> seems to be correct. New York City, NY, will never be "including black physicians." His remark conveys the impression that when Harriet Tubman<sup>2</sup> freed the slaves, this one would be a black physician. The next physician to be freed into a reservation is likely to be a Native American. This is analogous to the famous "separate but equal" applied to our education system.

Having asked "Will the poor and minorities be able to access to private practitioners?" he therefore encouraged minorities to go to public clinics and/or seek "nurse practitioners." He no longer "separate but equal" but a more pragmatic "some care is better than none." Soon we would be knocking on doors to the health side door for the physician.

In either case, Dr Ginzberg is recommending a drastic change in the principles of equality. Whatever new physician system is set up, it should provide access to quality care.

1. Ginzberg E: A new physician shortage. *JAMA* 1983;250:2621-2622.

In Reply.—I share with the letter writer a deep dissatisfaction with the fact that my colleagues and I have to refer to quality care, referring to realities rather than anybody else's preferences. Interestingly, despite the lack of physicians entering the field, I see little likelihood of physicians setting up pri-

2023280231

### Results

The levels of breath CO relative to ambient CO found in the population studied are shown in fig 1 for the 99 nonsmokers and in fig 2 for the 69 smokers (of whom 66 were cigarette smokers). The breath CO levels of the smokers were in general higher than those of the nonsmokers, ranging from 3 to more than 100 ppm, only 6% of smokers having breath CO levels lower than 6 ppm and 74% having levels above 10 ppm. Although most (79%) of the nonsmokers had levels below 6 ppm, 12% had levels above 10 ppm. The range for the nonsmokers was 2-60 ppm.

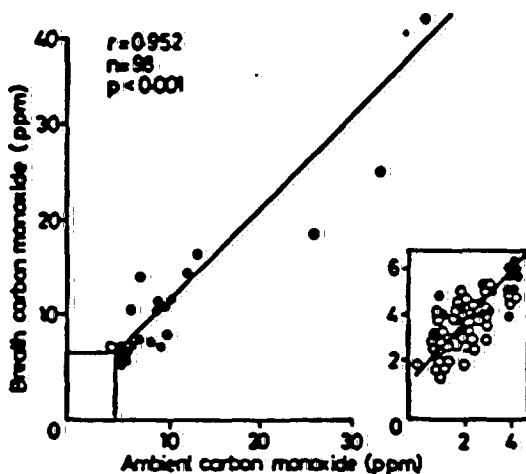


Fig. 1 Relation between breath and ambient (room) carbon monoxide levels in nonsmokers, in rooms with non CO generating heating (○) and possible CO generating heating (●), with calculated regression line.

Ambient CO levels in the respondents' homes were found to range from 0 to 42 ppm. The higher ambient CO levels were found in rooms where certain types of heating were operating: radiant gas fires (particularly if the gas fire was turned down low and was of the type where the element was then no longer incandescent), open coal or wood fires, coal or wood stoves, and paraffin heaters. Rooms being heated with these types of heating were designated CO generating (fig 3). Low ambient CO levels were almost always found in rooms where there was no heating on, or where the heating was by water or oil filled radiator or electric fire—non CO generating heating (fig 3). In nonsmokers, there was a very close correlation ( $r = 0.952$ ,  $p < 0.001$ ) between the

B D Cox and Margaret J Whichelow

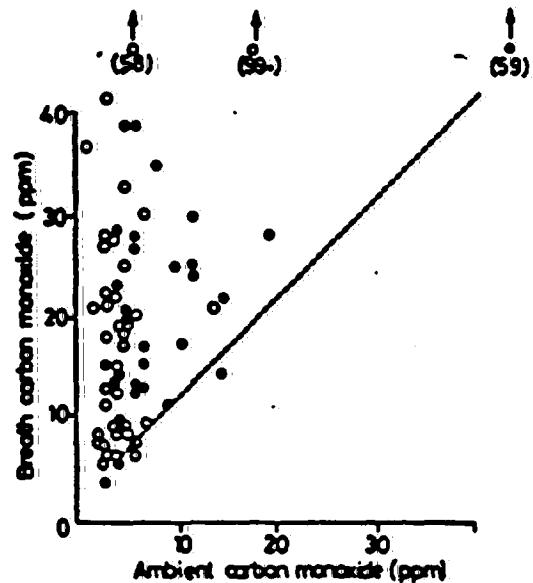


Fig. 2 Relation between breath and ambient (room) carbon monoxide levels in smokers, in rooms with non CO generating heating (○) and possible CO generating heating (●), compared with the regression line for nonsmokers.

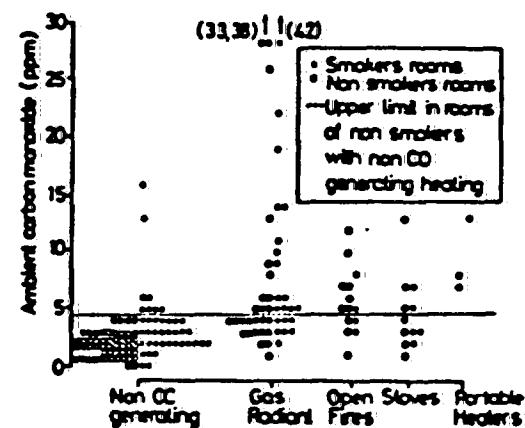


Fig. 3 Levels of ambient carbon monoxide in rooms of smokers (●) and nonsmokers (○) with various types of heating. "Non CO generating" includes rooms with no heating operating.

2023380232

## Carbon monoxide levels in the breath of smokers and nonsmokers: effect of domestic heating systems

B D COX AND MARGARET J WHICHELOW

*From the Office of the Regius Professor of Physic, Cambridge University School of Clinical Medicine, Addenbrooke's Hospital, Hills Road, Cambridge CB2 2QQ*

**SUMMARY** Breath and ambient (room) carbon monoxide (CO) levels were measured in a random sample of 168 adults in their own homes. The levels of breath CO in the 69 smokers ranged from 3 ppm to over 100 ppm, 74% being above 10 ppm; mean levels in the 99 nonsmokers were lower than in the smokers, 79% being below 6 ppm. In the remaining 21% of nonsmokers with higher breath levels than expected, the ambient CO was also found to be elevated, ranging up to 38 ppm. A close correlation in the nonsmokers was found between the breath and ambient CO levels ( $r = 0.952$ ,  $p < 0.001$ ). The rooms with the elevated ambient CO levels (above 5 ppm) were those which, at the time of testing, were being heated by gas radiant heaters, open fires or stoves. The maximum ambient CO in the rooms of smokers with non CO generating heating was 16 ppm. The results suggest that many people, both smokers and nonsmokers, may be at risk from CO generated by certain domestic heating systems and that nonsmokers are far more likely to be exposed to high levels of CO from these sources than from being in a room with a heavy smoker. Poor ventilation associated with the current trend towards excluding all draughts is likely to exacerbate the situation for both smokers and nonsmokers.

As a precursor to a nationwide study of various factors affecting health and health attitudes, two pilot studies were carried out on a random sample of adults. Included in these surveys, which were conducted in the subjects' own homes, were measurements of breath carbon monoxide. Carbon monoxide (CO) levels in breath correlate well with carboxyhaemoglobin in the blood<sup>1,2</sup> and have been used as an indicator of a smoker.<sup>1,2</sup> In the present studies, it was planned to use the breath CO measurements as a check on the subjects' reported smoking habits. It soon became evident that the simultaneous measurement of environmental CO was also necessary, when the levels of CO in the breath of some nonsmokers far exceeded the anticipated low values.<sup>3,4</sup> This may be important in view of the government's recent decision to publish the CO content of cigarettes, and the current interest in the possible health hazards of chronic CO exposure.<sup>5</sup>

### Methods

The subjects comprised 86 men and 82 women chosen at random from the electoral registers of

Bristol, Avon, and Bury St Edmunds, Suffolk. Their ages ranged from 18 to 74 years. The selections were made from electoral wards which had a distribution of socioeconomic groups similar to that existing in the United Kingdom as a whole.

The equipment used for the measurement of CO was a battery powered portable Ecolyzer (supplied by Analysis Automation Ltd) which will detect CO levels down to a concentration of less than one part per million.

The estimations were made in the subjects' own homes in the room in which the interview was being conducted (usually the sitting room).

The type of heating system in operation was noted. A measurement of the ambient CO level was made and after this the subject took a deep breath and held it for 20 seconds (to allow for equilibration with the CO concentration in the blood) and then blew into a trilaminated plastic bag which was subsequently attached to the sampling port of the Ecolyzer. The CO concentration in the expired air was measured and recorded. If the subject was a smoker, the time of the last cigarette and the number of cigarettes smoked per day were noted.

2023380233

ambient CO and the breath CO (fig 1). The regression coefficient of 1.03 shows that for a rise of 1 ppm CO in the atmosphere there was an almost equal rise of CO in the breath of nonsmokers. The regression intercept with the abscissa indicates that breath CO levels are 1.5 ppm higher at all levels than the ambient. The 1.5 ppm CO difference reflects *in vivo* metabolic production of CO.<sup>8</sup> The nonsmoker with the highest breath CO of 60 ppm in an ambient atmosphere of 3 ppm was excluded from the analysis as he was a welder using gas welding equipment who had just arrived home from work.

A significant relation between the breath and ambient CO levels in the smokers was also found ( $r = 0.430$ ,  $p < 0.001$ ) (fig 2). Inspection of the regression line of fig 2 reveals that for any one level of ambient CO the breath level was usually much higher than in the nonsmokers. However, when the smokers in rooms with potential CO generating heating appliances were excluded from the analysis, there was no correlation between breath and ambient CO ( $n = 37$ ,  $r = 0.058$ , NS).

Although it is apparent that the type of heating can markedly influence the ambient CO in a home, analysis of the results in environments where non CO generating heating devices were in operation showed that smokers do also contribute to the ambient CO. In the non CO generating rooms of the nonsmokers, there were no values above 4 ppm, whereas in similar rooms of smokers 21% of the values were above 4 ppm with a range of 0–16 ppm, and this difference was significant for the rooms of all smokers ( $\chi^2 = 4.613$ ,  $p < 0.05$ ) and particularly for those of heavy smokers (20 or more cigarettes per day) compared with those of nonsmokers ( $\chi^2 = 12.45$ ,  $p < 0.001$ ).

There was a significant correlation between the CO levels in the breath of smokers in a non CO generating environment and their reported daily consumption of cigarettes ( $n = 37$ ,  $r = 0.582$ ,  $p < 0.01$ ). However, a similar relation did not exist among smokers in rooms where potential CO generating heating systems were in operation ( $n = 31$ ,  $r = 0.033$ , NS). These smokers were found to have significantly elevated breath levels in relation to their daily consumption of cigarettes compared with the previous group ( $t = 2.4145$ ,  $p < 0.025$ ), thus indicating that heating systems and cigarette smoking were both contributing to their breath CO.

#### Discussion

Although it has been found<sup>1,8</sup> that the measurement of expired breath CO is a useful way of discriminating between smokers and nonsmokers, these previous studies were carried out on subjects undergoing a

medical examination at a BUPA clinic where the ambient CO level was no higher than the normally expected value of 3 ppm.<sup>9</sup> It can be assumed that a considerable period of time had elapsed between the subjects leaving home or their place of work and being tested at the clinic, and that therefore equilibration between the carboxyhaemoglobin and the ambient CO at the clinic had taken place.<sup>9</sup> In the present study, however, measurements took place in the living rooms of the respondents' homes, where many of the ambient CO levels far exceeded the expected ceiling level of 3 ppm. Although a proportion of the excess CO was, in the case of smokers, contributed by their own cigarettes (fig 2), a far greater amount was contributed by the CO generating heating systems. It must be acknowledged that some of the excess CO may have come from other (particularly heavy) smokers in the household, but data were not collected in these pilot surveys on the smoking habits of other members of the household.

The observation that radiant gas fires, open coal and wood fires, coal and wood stoves, and portable paraffin and gas heaters in use in the room at the time the CO measurements were made were associated with elevated CO levels is particularly disturbing in view of the trend back towards open fires and stoves, and of the current emphasis on double glazing and efficient draught exclusion. Without adequate ventilation, the products of combustion remain in the room. High levels of CO indicate that combustion was incomplete, and observations at the time the measurements were made revealed that with the radiant gas fires, at least, the ambient CO levels were highest when these appliances were turned down low, particularly if they were of the older type where the elements, when the fire is turned down, are no longer incandescent.

It has been suggested that moderately elevated blood carboxyhaemoglobin levels, an average of 5.1%,<sup>10</sup> derived from cigarettes or the environment are associated with disease.<sup>10</sup> The breath CO levels in the nonsmokers in this study in rooms with CO generating heating operating, ranging from 6 to 42 ppm, would be expected to reflect carboxyhaemoglobin levels from about 1% to 7% in these subjects. This suggests that such nonsmokers may also be at risk from cardiovascular disease and indicates that means should be found to ensure complete fuel combustion or more efficient ventilation of heating appliances. Although extensive double glazing and draught proofing have been introduced to conserve heat, in Sweden this has been accompanied by fitting ducted ventilation systems, forcing the escaping warm air over heat exchangers to heat the incoming air.

2023380234

We are grateful to our colleagues in the Office of the Regius Professor of Physic, Dr M Wadsworth, and medical students of the University of Bristol for their assistance in the collection of the data.

• References

- <sup>1</sup>Cohen SI, Perkins NM, Ury HK, Goldsmith JR. Carbon monoxide uptake in cigarette smoking. *Arch Environ Health* 1971; 22: 55-60.
- <sup>2</sup>Wald NJ, Idle M, Boreham J, Bailey A. Carbon monoxide in breath in relation to smoking and carboxyhaemoglobin levels. *Thorax* 1981; 36: 366-9.
- <sup>3</sup>Cole PV. Comparative effects of atmospheric pollution and cigarette smoking on carbon-haemoglobin levels in man. *Nature* 1975; 235: 699-701.

- <sup>4</sup>Borland C, Chamberlain A, Higenbottam T, Shipley AL, Rose G. Carbon monoxide yield of cigarettes and its relation to cardiorespiratory disease. *Br Med J* 1983; 287: 1583-6.
- <sup>5</sup>Ringold A, Goldsmith JR, Helwig HL, Finn R, Schuette F. Estimating recent carbon monoxide exposures. *Arch Environ Health* 1962; 5: 308-18.
- <sup>6</sup>Clyne C, Arch PJ. Nicotine, carbon monoxide and heart disease. *Lancet* 1961; ii: 1044-5.

2023380235

# PONSE

## CO<sub>2</sub> Concentrations

### Editor:

The following comments should have been included in our discussion paper (September 1982, JAPCA, p. 918) about John Yocum's "Indoor-outdoor air quality relationships: a critical review" (May 1982, JAPCA, p. 500). Yocum's review contained two errors which should be corrected.

He states on page 508 "In this same work, the LBL workers showed that sidestream cigarette smoke produced 130 µg CO/mg of tobacco burned. This value is about half that reported in the NAS report." In fact the NAS report *Indoor Pollutants* (1981) National Academy Press, Washington, D.C. lists 86.3 mg CO per cigarette in sidestream cigarette smoke and 758 mg tobacco per cigarette for an emission rate of 114 µg CO/mg tobacco burned. Thus our work<sup>1</sup> and that cited in the NAS report are in very good agreement. (If the values of 86.3 mg CO per cigarette and 411 mg tobacco burned only during the production of sidestream smoke are used, the emission rate of 210 µg CO/mg for tobacco burned only during the production of sidestream smoke is obtained. As this rate is about double our measured rate, we suggest this may have caused Yocum's error).

Yocum also states that no studies clearly show the specific effect of smoking on I/O ratios for CO in normally occupied residences. However, the same study<sup>2</sup> he cited to show the effect of cigarette smoking on RPM levels in a house equipped only with electrical appliances also demonstrated the effect of cigarette smoking on CO levels in a normally occupied residence. In this study CO concentrations during the period when cigarettes were smoked were only 0.1 to 0.2 ppm higher than the period when no smoking occurred. This is identical with the CO concentration increase one would predict by using the increase in particulate levels observed and the ratio of the mass of CO produced to the mass of RSM produced as derived from LBL's measured rates.

This strongly suggests that CO is a minor (or negligible) pollutant in sidestream cigarette smoke as compared to RPM emissions—for example, an increase in CO of 1 ppm would correspond to an increase in RPM level of 154 µg/m<sup>3</sup>, thus dominating IAQ. Based on theoretical and empirical results, CO sidestream emissions from cigarettes have often been overemphasized.

John R. Girman and Greg W. Traynor, Staff Scientists, Building Ventilation and Indoor Air Quality Program, Lawrence Berkeley Laboratory.

## References

1. J. R. Girman, M. G. Apt, G. W. Traynor, C. D. Hollowell, "Pollutant Emission Rates from Indoor Combustion Appliances and Sidestream Cigarette Smoke," presented at the International Symposium on Indoor Air Pollution, Health and Energy Conservation, University of Massachusetts, Amherst, MA, October 1981, Lawrence Berkeley Laboratory Report LBL-12562.
2. J. V. Berk, R. Young, S. R. Brown, C. D. Hollowell, "Impact of Energy Conserving Retrofits on Indoor Air Quality in Residential Housing," presented at the 74th Annual Meeting of the Air Pollution Control Association, Philadelphia, PA, June 1981, Lawrence Berkeley Laboratory Report LBL-12189.

### Author's reply

I appreciate the comments by Girman and Traynor on my recent Critical Review. They are correct in asserting that I should have used the total tobacco burned from the NAS figures rather than only that burned during sidestream smoke production in order to compare these figures with those developed by LBL.

The second point deals with my statement on the lack of studies clearly showing the effect of smoking on indoor CO concentrations "in normally occupied residences." In the LBL work cited, House No. 1 had one occupant who "was a cigarette smoker who consumed 20–40 cigarettes per day." It is questionable that the 0.1 to 0.2 ppm increase in CO concentration between smoking and non-smoking periods can be relied upon as a quantitative indication of the effects of cigarette smoking in view of the wide range of possible smoking rates and standard deviations of the same order as the average concentrations. Nevertheless, I will agree that contributions to indoor levels of CO by smoking in "normally occupied residences" are probably quite small.

John E. Yocom, TRC Environmental Consultants, Inc., East Hartford, CT.

## Opacity Measurements

### Editor:

A recent paper by William D. Conner and Harold B. McElhoe<sup>1</sup> provides useful data indicating good agreement between opacity measurements by visual observation and measurements obtained with an optical instrument mounted in a stack. Since measurement and control of particulate matter is an important subject in the air pollution control field, some general comments on the use of these two measurement techniques might be appropriate.

This paper adds to a considerable body of information that goes back to

the original publication by Maximilian Ringelman in 1895.<sup>2</sup> In fact, William Conner was a coauthor of a useful manual published in 1967 by the U.S. Public Health Service.<sup>3</sup> This manual was used extensively by many governmental agencies in establishing legal regulations and methods of implementation.

So far as I am aware, the first legal regulation incorporating in-stack transmissometer measurements was adopted by the Texas Air Control Board in 1969.<sup>4</sup> Experience has demonstrated that the instrumental problems can be solved to provide a practical method of control for use both by industries subject to control and by governmental agencies responsible for surveillance and enforcement.<sup>5,6</sup> Such a regulation has many advantages compared to a regulation based on visual observation, for reasons outlined in the references cited.

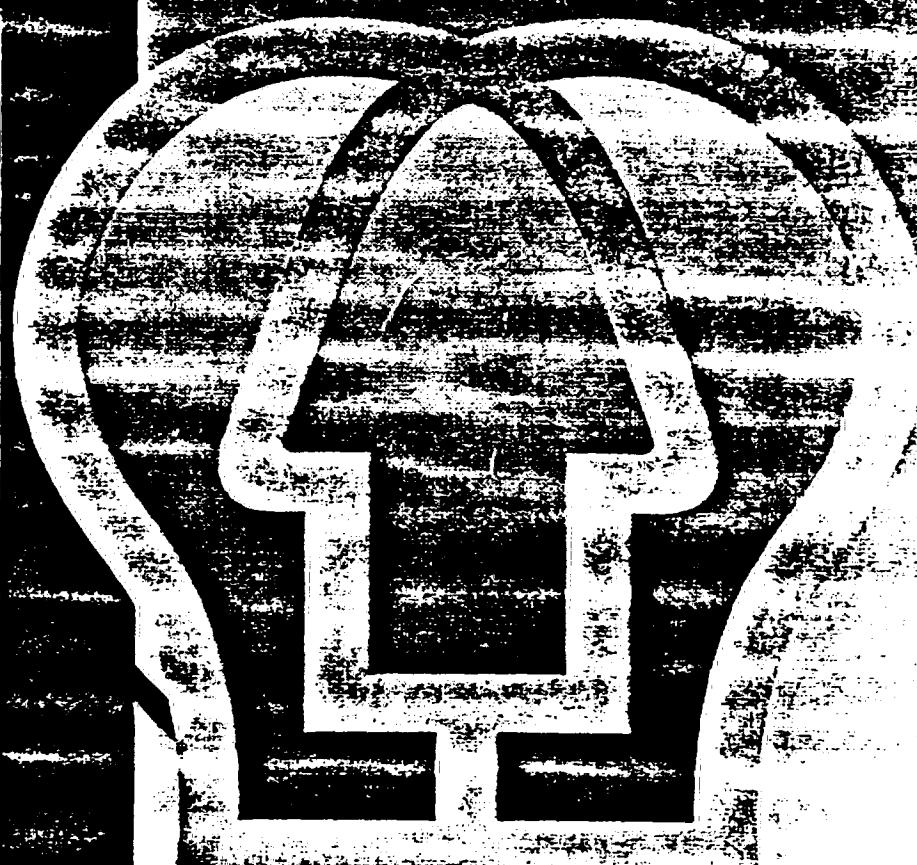
While this study<sup>1</sup> and others have demonstrated that instrumental measurements and visual observations usually agree within reasonable limits, such agreement is not essential for transmissometer measurements to be useful. The two techniques are not measuring exactly the same thing, so some occasional differences are not surprising. Neither technique is intrinsically "correct," so a difference does not necessarily mean that one is right and the other wrong. As pointed out by Conner and McElhoe, some of the differences are caused by the fact that visual observations are adversely affected by conditions such as darkness or cloud cover, in which case the instrumental measurement is more reliable. The Texas regulation avoided problems with any possible differences by providing that a company would not be legally responsible for a limit based on visual observations if a transmissometer was installed and calibrated according to the specified procedure and continuous monitoring data made available to a governmental control agency on request.

While this paper dealt with measurement techniques rather than basic principles and underlying philosophies of emission control, they infer the same philosophy that has been used in the past, i.e., that mass emission limits should be the primary basis for control, while opacity is useful as a quick means of determining a possible violation of the mass emission limit. I suggest that this philosophy should be questioned on the basis of variations in particle size, and that mass emission limits and opacity limits are both valid on their own merits.

Biomedical research workers have established over the past several decades that the particle size range most likely

(Continued on page 90)

THE FIFTH  
INTERNATIONAL  
CONFERENCE  
ON INDOOR AIR  
QUALITY AND  
CLIMATE



INDOOR AIR '90

2023380237

## **MEASUREMENTS OF INDOOR CARBON MONOXIDE LEVELS USING PASSIVE SAMPLERS IN KOREA**

**H. Nitta, B.-S. Son, K. Maeda**

Department of Epidemiology, School of Health Sciences, University of Tokyo  
Bunkyo-ku, Tokyo 113 JAPAN

**Y.-S. Kim**

Department of Medical Information and Management, College of Medicine, Hanyang University Sungdong-ku, Seoul 133, KOREA

**Y. Yanagisawa**

Department of Environmental Science and Physiology, Harvard School of Public Health Boston, MA 02115, U.S.A.

Indoor carbon monoxide (CO) concentrations and personal CO exposures were measured in Korea where CO poisoning caused from the usage of Yeontan or coal briquette as domestic fuel for cooking and space heating has been a serious problem. Thirty-five homes were selected from an urban and rural area for the survey conducted in January 1989. Newly developed passive CO samplers were placed in a kitchen and living room for the indoor measurement and were worn by a housewife for the personal exposure monitoring. Daily averages of indoor CO concentrations were 23.4 ppm in the kitchen and 11.8 ppm in the living room. The average personal CO exposures of 18.1 ppm was between the two indoor CO concentrations. The indoor concentrations and personal exposures to CO were different in types of the space heating systems and two areas. House ventilating methods and socio-economic conditions were also important factors in determining the indoor and personal CO levels in Korea.

### **INTRODUCTION**

One hundred thousand poisoning cases and two thousand deaths in a year due to accidental exposures to CO are reported in Korea<sup>1</sup>, where approximately 70 percent of households use coal briquettes or Yeontan as domestic fuel for cooking and space heating. CO, a combustion product of Yeontan, is discharged into a house through flues of the heating system which is inadequately maintained and/or installed. An ondol is a traditional and common method for floor heating and cooking in Korea. There are two basic types of ondols using Yeontan. One is a traditional ondol and the other is an ondol boiler. In the traditional ondol, flues are installed from the cooking stoves in kitchens, under floors, through the living area, to the outside. Indoor

2023380238

2023380239

air is warmed up with the combustion gas vented through the flues. In the ondol boiler, hot water or steam made with the boiler is used instead of the combustion gas as the medium for the floor heating. The pipes for the hot water or steam are positioned under the floors. "Saemaul" boiler is a subtype of the ondol boiler using a cooking stove for producing hot water.

Since most of the research has focused on medical treatments and follow-up studies on the patients suffered from the CO poisoning, there are few studies on personal CO exposures and indoor CO concentrations. A preliminary field study using newly developed passive CO monitors was conducted to investigate the distribution of personal CO exposures and to seek determinant factors of indoor CO pollution in Korea.

#### MATERIALS & METHODS

Thirty five participants in two cities, Seoul and Togo, were selected from homes using Yeontan. Seoul is the largest city in Korea and Togo is a rural area located at about 130 km south of Seoul. Twenty five participants were chosen from three districts in Seoul where socio-economic conditions differed.

The sampling was conducted, in January 1989, using three passive CO samplers in each home for 24 hours. Two samplers for the indoor measurements were placed in a kitchen and living room. One sampler for monitoring the personal exposure was carried by a housewife. Some outdoor samplers were placed at the outside of several participant's houses. CO levels inside and outside of each house were monitored with a portable electrochemical CO analyzer prior to the passive sampler measurements in order to determine appropriate sampling points.

The passive CO sampler consisted of a glass tube with one side sealed by a rubber cap<sup>3</sup>. The CO adsorbent packed in the glass tube was made from Zn-Y-zeolite. The sampling rate of CO was controlled with a narrow diameter polyethylene tube inserted into the center of the adsorbent layer through a septum fixed to the other end of the glass tube. Analysis of the samplers was carried out by thermal desorption of CO, followed by gas chromatography with a flame ionization detector.

Participants' activities were recorded during the measurements, such as the time spent, locations, and the usage of gas appliances. We also collected information on participant's smoking habits and house characteristics.

#### RESULTS & DISCUSSION

Characteristics of house structures and types of ondols of 35 homes are summarized Table 1. There are more Korean style homes in Seoul, which were less spacious and mostly made of wood. Ten homes have the traditional

ondol, 10 use the Saemaul boiler and the rest are users of the ondol boiler. In Togo, 2 homes have the traditional ondol and 8 use the ondol boiler.

Cumulative frequency distributions for indoor CO concentrations and personal exposure levels are presented in Figure 1 and a summary of statistics is shown in Table 2. The mean CO concentrations in the kitchen and living room were 23 ppm and 12 ppm, respectively. The average personal CO exposure of 18 ppm was between the averages of the two indoor measurements. Half of the participants were exposed to high levels of CO exceeding the ambient air quality standard in the USA and Japan which is a 24-hour average of 10 ppm. Average outdoor CO concentrations measured by the electrochemical analyzer were 5.5 ppm in Seoul and 1.3 ppm in Togo.

When comparing indoor CO concentrations by types of ondols, houses with the ondol boiler had lower mean concentrations than houses using the traditional ondol. Living room CO concentrations in Seoul, for example, were 11.1 ppm and 9.5 ppm for the traditional ondol and the ondol boiler respectively (Table 3). In Togo, they were 14.2 ppm and 4.7 ppm (Table 4). Houses with the Saemaul boiler were the most polluted by CO among the three ondol systems in Seoul (Table 3). The Saemaul boiler system, which has been recently adopted, does not help to improve indoor air quality in terms of CO.

The average personal CO exposures were highest for the wives using the traditional ondol in Togo, while in Seoul the average CO levels were highest among wives using the Saemaul boiler (Table 3 & 4). When comparing averages of the personal CO exposures by the type of ondols and by areas, they did not necessarily correspond to the extent of indoor air pollution by CO. In Seoul, the mean personal CO exposures of the traditional ondol users and the ondol boiler users were 11 ppm and 19 ppm, whereas indoor CO concentrations in the traditional ondol houses were higher than those in the ondol boiler houses. These suggest that the personal CO exposures depend not only on the indoor CO concentrations but also on other factors, such as daily activities, outdoor CO concentrations and house ventilation.

Presence of smokers in homes was not a major determinant factor of the CO concentrations (Table 5). We could not detect the contributions of smoking to indoor CO levels and personal exposure.

#### CONCLUSIONS

The types of heating systems were found to be one of the determinant factors of indoor CO concentrations and personal CO exposures. Indoor CO levels for homes with traditional ondols were higher than those with ondol boilers in both areas. Particularly kitchen CO concentrations of the traditional

2023380240

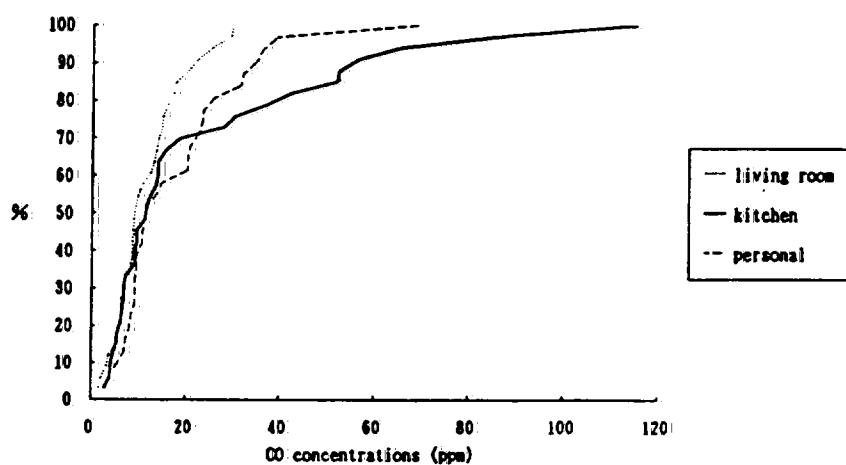
ondol house in Togo exceeded 50 ppm. In Seoul the houses with the Saemaul boiler, which is a subtype of the ondol boiler, had the highest indoor CO concentrations. The personal CO exposures were between indoor CO concentrations in the living room and kitchen in most cases.

#### REFERENCES

1. Song DB (1985) Epidemiology of Carbon monoxide poisoning in Korea. J of Korean Med Assoc, 28:1059-1063.
2. Hishinuma M, Yanagisawa Y (1989) Passive sampler for carbon monoxide using a solid adsorbent. The 82nd Annual Meeting of Air Pollution Control Association, Anaheim, CA.

Figure 1.

Cumulative frequency distributions of CO concentrations by location.



2023380241

TABLE 1. CHARACTERISTICS OF SUBJECTS' HOUSE BY THE AREA.

	SEOUL	TOGO
	# OF HOUSES	# OF HOUSES
<b>TYPE OF HEATING:</b>		
TRADITIONAL ONDOL	10(40%)	2(20%)
ONDOL BOILER		
SAEMAUL	10(40%)	0( 0%)
OTHER	5(10%)	8(80%)
<b>STRUCTURE</b>		
KOREAN STYLE	16(64%)	2(20%)
WESTERN STYLE	2( 8%)	4(40%)
KOREAN-WESTERN	7(28%)	4(40%)
MIXED STYLE		
	MEAN(S.D.)	MEAN(S.D.)
TOTAL FLOOR SPACE*	37.3(29.4)	80.9(36.6)
KITCHEN SPACE*	5.3( 4.0)	4.0( 2.0)
LIVING ROOM SPACE*	6.9( 3.3)	5.3( 1.7)

\*SQUARE METER

TABLE 2. SUMMARY STATISTICS OF CO CONCENTRATIONS. (PPM)

	MEAN	S.D.	RANGE	N
LIVING ROOM	11.8	7.2	1.7 - 29.8	33
KITCHEN	23.4	26.2	2.8 - 115.1	33
PERSONAL	18.1	13.8	3.0 - 69.5	31

2023380242

TABLE 3. MEAN CO CONCENTRATIONS (PPM) BY TYPE OF HEATING IN SEOUL.

	TRADITIONAL ONDOL	ONDOL BOILER SAEMAUL	OTHERS
LIVING ROOM	11.1 (N=10)	18.1 (N=10)	9.5 (N=4)
KITCHEN	16.7 (N=9)	29.5 (N=10)	11.4 (N=4)
PERSONAL	10.8 (N=10)	25.0 (N=9)	18.9 (N=3)

TABLE 4. MEAN CO CONCENTRATIONS BY TYPE OF HEATING IN TOGO.

	TRADITIONAL ONDOL	ONDOL BOILER
LIVING ROOM	14.2 (N=2)	4.7 (N=7)
KITCHEN	57.6 (N=2)	21.0 (N=8)
PERSONAL	50.6 (N=2)	10.0 (N=7)

TABLE 5. MEAN CO CONCENTRATIONS (PPM) BY PRESENCE OF SMOKERS IN HOMES.

	SMOKERS	
	YES (24)*	NO (11)*
	MEAN (S.D.)	MEAN (S.D.)
LIVING ROOM	12.6 ( 8.0)	10.3 ( 4.8)
KITCHEN	23.1 (26.1)	24.1 (26.6)
PERSONAL	15.4 ( 8.5)	23.7 (19.8)

\* (NUMBER OF DATA)

2023380243

development programs are underway through sponsorship of the U.S. Department of Agriculture, National Science Foundation, Southwest Border Regional Commission, Four-Corners Regional Commission, and the state of California.

Germ plasm collections have been made from wild guayule plants in Mexico and Texas. Plantings have been established to test yields, to increase seed supplies, and to conduct plant breeding work. Test plots have been established to determine desirable planting and cultivating practices. Research is being conducted on the possibility of increasing rubber yield by treating guayule plants with plant growth regulators.

The recent development of a seed coating process to promote germination, and the development of selective herbicides, will make direct seeding in field plantations a possibility. Eliminating nursery or greenhouse propagation could produce considerable savings in production costs.

The only guayule yield figures now available are estimates developed during the ERP. During the life of the ERP the

1800 hectares that were planted yielded, per hectare, approximately 480 kg of guayule rubber per year. Kelly (15) obtained yields of approximately 860 kg per hectare per year from one test plot in California. Foster et al. (16) have outlined the state of the art of guayule technology and described present and projected world rubber market conditions and areas of the United States where conditions favor guayule cultivation.

#### References and Notes:

1. W. G. McGinnies, B. J. Goldman, P. Payne, Eds., *Food Fiber and the Arid Lands* (Univ. of Arizona Press, Tucson, 1971); J. D. Johnson, *Arid Lands Newsletter*, No. 6 (1977); \_\_\_\_ and E. Cruz-Turbe, *Proceedings of IV Symposium Sobre El Medio Ambiente del Golfo de California, Mazatlán, Sinaloa, México, 9 to 12 May 1979* (in press); R. S. Feiger, in *New Agricultural Crops*, G. A. Ritchie, Ed. (Westview Press, Boulder, 1979), p. 5.
2. W. P. Bemin, J. W. Berry, C. W. Weber, *Arid Lands Newsletter*, No. 8 (1978); in *New Agricultural Crops*, G. A. Ritchie, Ed. (Westview Press, Boulder, 1979), p. 63.
3. L. B. Waymack et al., paper presented at Arizona Cattle Feeders' Day, University of Arizona, Tucson, 1 to 4 September 1976.
4. J. Hoffmann, personal communication.
5. W. C. Sherbrooke and E. F. Haase, *Jojoba: A Wax-Producing Shrub of the Sonora Desert* (Office of Arid Lands Studies, University of Arizona, Tucson, 1974); W. C. Sherbrooke,
6. Jojoba Happenings (Newsletter) No. 20 (1979).
7. National Research Council, *Products from Jojoba: A Promising New Crop for Arid Lands* (National Academy of Sciences, Washington, D.C., 1973).
8. San Carlos Jojoba Project, P.O. Box 681, San Carlos, Ariz. 85350.
9. D. M. Yermanos, *Calif. Agric.* 33, 718 (1979).
10. L. Hogan, in *New Agricultural Crops*, G. A. Ritchie, Ed. (Westview Press, Boulder, 1979), p. 177.
11. J. D. Johnson, *Jojoba Happenings (Newsletter)*, No. 27 (1979).
12. M. Calvin, *Chem. Eng. News* 56 (No. 12), 30 (1978); P. E. Nielsen, H. Nishimura, J. W. O'Brien, M. Calvin, *Science* 198, 342 (1977).
13. R. A. Buchanan, J. M. Culb, F. H. Orey, C. R. Russell, in *Proceedings of the 110th Meeting of the Rubber Division, American Chemical Society, San Francisco, 3-8 October 1976*.
14. National Academy of Sciences, *Guayule: An Alternative Source of Natural Rubber* (National Academy of Sciences, Washington, D.C., 1977).
15. Comisión Nacional de las Zonas Áridas, Subdirección Industrial, *Informe de Actividades, 1972-1976* (Comisión de las Zonas Áridas, Mexico, D.F., 1976).
16. O. J. Kelly, in *An International Conference on the Utilization of Guayule*, Proceedings, W. G. McGinnies and E. F. Haase, Eds. (Office of Arid Lands Studies, University of Arizona, Tucson, 1975), p. 83.
17. E. F. Foster et al., *A Socioeconomical Survey of Guayule Rubber Commercialization* (Midwest Research Institute, Kansas City, and Office of Arid Lands Studies, University of Arizona, Tucson, 1979). See also W. G. McGinnies and J. Taylor, *An Integrated Approach to Guayule Research* (Office of Arid Lands Studies, University of Arizona, Tucson, 1979).
18. The Diamond Shamrock Corporation, Cleveland, Ohio, is sponsoring the hydrocarbon research program at the University of Arizona.

## Indoor Air Pollution, Tobacco Smoke, and Public Health

James L. Repace and Alfred H. Lowrey

Serious health effects from air pollution have led to federal standards for the regulation of outdoor exposure levels. However, Americans spend about 90 percent of their time indoors (1). Thus the levels of indoor air pollution are important in determining total exposure to air pollutants (2-6). Indeed, in a recent review article (6) it was concluded that indoor air pollution in public office buildings is of greater potential harm than the outdoor variety, and that these exposures may constitute a real threat to the health of many urban people. The U.S. Surgeon General asserted in his report on *Smoking and Health* that tobacco smoke can be a significant source of atmospheric pollution in enclosed areas (7). Some 53 million U.S. smokers

consumed 615 billion cigarettes in 1978 (8). Thus it is apparent that indoor air pollution from tobacco smoke is pandemic.

In the presence of cigarette smoke, many normal nonsmokers experience eye and throat irritation, headache, rhinitis, and coughing; allergic persons report wheezing, sneezing, and nausea as well. Particularly acute symptoms may be found in infants, children, persons with cardiovascular or respiratory disease, and wearers of contact lenses (7, 9). Determining the extent of the exposure of nonsmokers to cigarette smoke is important because smoking is a cause of chronic obstructive pulmonary disease, cardiovascular disease, and lung cancer, and is associated with cancers in

other parts of the body (7); because these diseases also occur in nonsmokers; and because the products of tobacco combustion have been detected in nonsmokers (10).

Although measurements of indoor carbon monoxide pollution from smoking are abundant (7), published reports of the exposure of the population to the particulate phase of ambient tobacco smoke are rare (7, 11-13). Furthermore, a comprehensive theory of the generation and removal mechanisms for tobacco particulates in naturally or mechanically ventilated habitable spaces has not been presented.

We therefore undertook a systematic study of the levels of respirable suspended particulates (RSP) in several common indoor environments in an attempt: (i) to determine the relation of these levels to the aerosol from tobacco smoking, (ii) to understand the effect of ventilation on tobacco smoke concentration.

James L. Repace is an environmental protection specialist at the Environmental Protection Agency, Washington, D.C. 20460, and a former research physicist at the Naval Research Laboratory, Washington, D.C. 20375. Alfred H. Lowrey is a research chemist at the Laboratory for the Structure of Matter, Naval Research Laboratory. This work was sponsored by the Prince George's Environmental Coalition and was performed by the authors in their private capacity. No official support or endorsement by the Environmental Protection Agency, the Naval Research Laboratory, or any other federal agency is intended or should be inferred.

2023380244

tions, and (iii) to develop a general model for estimating the range of the public's exposure. Our goal was to provide a quantitative basis for assessing the health hazards to nonsmokers posed by repeated exposure to tobacco combustion products.

### Model Development.

To relate the contribution of smoking to indoor RSP requires a model describing the behavior of the tobacco aerosol in indoor spaces. Bridge and Corn (6) found that a reduced form of an equation by Turk (14) reliably predicts carbon monoxide (CO) concentrations from tobacco smoke in ventilated spaces and so is of major value in assessing the possible hazards in occupied spaces (11). The equation is not valid, however, for a pollutant that is affected by physical decay due to adsorption on room surfaces. Penkala and DeOliviera (15) showed that decay of the tobacco aerosol in a well-mixed unventilated chamber is exponential.

We modify the Turk equation in differential form by adding a decay term to the removal rate and equating the rate of change of pollutant mass to the algebraic sum of the generation and removal rates:

**Table 1. Recommended values of the mixing factor  $m$ , after Corn (11).** The mixing factor is an empirical number that accounts for room-specific effects on pollutant transport. Pollutant removal is more rapid in a well-mixed atmosphere (where  $m$  is large) than in a poorly mixed, stable one (where  $m$  is small). Factors that affect  $m$  include type and placement of ventilation grills, ventilation flow rates, inhomogeneous pollutant distribution, barriers, circulation fans, and room traffic.

Configuration of air supply system	$m$
Perforated ceiling*	1/2
Trunk system with anemostats	1/3
Trunk system with diffusers	1/4
Natural draft and ceiling exhaust fans	1/6
Infiltration and natural draft	1/10

\*This is the best standard condition.

decay time, a time constant associated with the removal of a pollutant from a room through adsorption on surfaces and filtration; and  $m$  is the mixing factor, an empirically determined number (16) that modifies the ventilation time as  $\tau_v/m$ , where  $m \leq 1$  ( $m = 1$  implies ideal mixing). Corn (11) suggested values of  $m$  for various ventilation systems (Table 1). We postulate that  $m$  also modifies the ideal decay time as  $\tau_a/m$ . The pollution generation rate, in micrograms per minute,

**Summary.** An experimental and theoretical investigation is made into the range and nature of the exposure of the nonsmoking public to respirable suspended particulates from cigarette smoke. A model incorporating both physical and sociological parameters is shown to be useful in understanding particulate levels from cigarette smoke in indoor environments. Observed levels of particulates correlate with the predictions of the model. It is shown that nonsmokers are exposed to significant air pollution burdens from indoor smoking. An assessment of the public health policy implications of these burdens is presented.

The solution yields the density  $A(t)$ , in micrograms per cubic meter, of smoke in the room as a function of time:

$$A(t) = A_{\infty}(1 - e^{-mt}) \quad (1)$$

where  $A_{\infty} = Gr/V$  is the equilibrium concentration of the pollutant in the room, and where the time constant

$$\tau = \frac{\tau_a \tau_v}{m(\tau_a + \tau_v)} \quad (2)$$

is the mean ventilation time, or the time for the smoke concentration to decrease to  $1/e$  of its value (where  $e$  is the base of natural logarithms);  $V$  is the room volume in cubic meters;  $\tau_v = V/Q$  is the ideal ventilation time, or the time required to replace a volume of air equal to the volume of the room by ventilation and infiltration;  $Q$  is the volume rate of ventilation and infiltration;  $\tau_a$  is the ideal

decay time, given as  $G = nC_0/\tau_a$ , where  $n$  is the number of cigarettes being smoked at time  $t$ ;  $C_0$  is the total particulate matter (TPM) from both sidestream and exhaled mainstream smoke; and  $\tau_a$  is the duration of cigarette smoking.

Equation 1 has two special cases: (i) in the case of ventilation only ( $\tau = \tau_v/m$ ) it becomes the reduced Turk equation of Bridge and Corn (6), with  $m = 1$ ; and (ii) in the case of adsorption only (the unventilated room),  $\tau = \tau_a/m$ . Then, if the generation of smoke ceases at time  $t_s$ , prior to equilibrium,  $A$  will decay according to

$$A(t) = A_0 e^{-mt''} \quad (3)$$

where  $A_0$  is a constant related to the equilibrium concentration by

$$A_0 = A_{\infty} [e^{mt_s} e^{\tau_a} - 1]$$

Equation 3 becomes the decay equation described by Penkala and DeOliviera (15) for  $m = 1$ .

The modified Turk equation (Eq. 1) contains only measurable quantities, and thus in principle can be used to estimate the concentration of TPM or CO from tobacco smoke (or other indoor air pollutants), as a function of time, for any room for which the pollutant generation rate, volume, and mean ventilation time are known.

### Controlled Experiments

Equation 2 shows that the mixing factor affects the time constant for decay as well as ventilation. Experiments under conditions of known ventilation were therefore necessary to assess the influence of mixing factors, decay time constants, and generation rates on the growth and decay of tobacco smoke particulates. To increase the usefulness of the experimental values determined for the mean ventilation time for the removal of tobacco smoke, we conducted these experiments in actual occupied spaces rather than in experimental chambers.

A piezobalance (TSI model 3500) (17-19) was used in sampling the aerosol. It collects respirable particulates (20) between 0.01 and 3.0 micrometers in diameter with near 100 percent efficiency (decreasing to 50 percent at 3.5  $\mu\text{m}$  and to 10 percent at 4  $\mu\text{m}$ ). The sampling rate is 1 liter/min (18); the sampling time is variable. Factory-calibrated with welding smoke, the detecting crystal in the instrument used has a sensitivity of 5.74  $\mu\text{g}/\text{min}\cdot\text{m}^2$  per hertz. The instrument underestimates the mass concentration of tobacco aerosol by about 15 percent compared to measurements made with low-volume filter sampling techniques. Readings can be affected by changes in humidity; the maximum expected error due to changes in relative humidity when sampling a hygroscopic aerosol (such as tobacco smoke) is given as  $\pm 10 \mu\text{g}/\text{m}^3$ . The overall instrument error is about  $\pm 10$  percent compared with low-volume filter measurements of welding smoke (19). The aerosol from sidestream cigarette smoke (that portion emitted by the burning tip), an important component of many indoor aerosols, is log-normal, with 99 percent of the mass  $< 1 \mu\text{m}$  in aerodynamic diameter and with an initial mass median diameter (MMD) from 0.2 to 1.5  $\mu\text{m}$  depending on dilution (20, 21). The relative particle sizes of fresh sidestream and mainstream smoke (the latter being that portion inhaled by the smoker) are about the same; for ex-

2023380245

Table 2. Parameters for Eq. 1, as determined with experiments 1 to 3 (unventilated rooms).

Experiment	$A_{in}$ ( $\mu\text{g}/\text{m}^3$ )	$A_{out}$ ( $\mu\text{g}/\text{m}^3$ )	$\tau_e/\text{min}$ (min)	$m$	$r^{20}$	$C_0$ (mg of TPM)	Cigarette condition
12	530	503	10	1	.98	12.3	Smoldered
22	5178	551	89	1/9	.42	16.0	Smoldered
38	1773	681	16.4	< 1	.81	23.0	Smoked

\*Coefficient of determination for the decay curve. <sup>†</sup>The estimated amount of TPM liberated if the entire cigarette had been consumed, according to FTC protocol. The FTC mainstream TPM level for this cigarette is 18 mg (24). <sup>‡</sup>V = 21.9 m<sup>3</sup>. <sup>§</sup>V = 29 m<sup>3</sup>.

haled mainstream smoke, particle size is estimated to be  $\sim 0.8 \mu\text{m}$  (MMD). Since the ambient cigarette smoke aerosol is reproducible and coagulates very slowly, it has been used as a test aerosol (21) and in evaluation of heating, air-conditioning, and ventilating systems (22). [The bulk of the ambient tobacco aerosol is probably due to cigarettes, since less than 15 percent of smokers smoke cigars or pipes (23).]

#### Unventilated Growth and Decay of Tobacco Smoke

Experiments were carried out to determine the usefulness of Eq. 3, which predicts a rapid decay for good mixing and a

slow decay for poor mixing; and also to discover the limits of  $\tau_e/\text{min}$ .

Experiments 1 and 2 were conducted in a wood-paneled den in a private residence. In the geometric center of the room (volume, 21.9 m<sup>3</sup>), a popular filter cigarette [containing 65 millimeters of tobacco and ranking 94th on the Federal Trade Commission (FTC) scale of tar and nicotine content (17 milligrams of tar and 1 milligram of nicotine) (24)] was ignited and allowed to smolder until 89 percent of its tobacco was consumed. During the first experiment, two box fans (31 centimeters in diameter) with anti-parallel exhausts were used to ensure ideal mixing; each fan's exhaust, measured with a Velometer, was 55 m<sup>3</sup>/min. The growth and decay of the RSP were

measured with the piezobalance. Experiment 2 was similar to experiment 1, except that the cigarette was extinguished after 75 percent of its tobacco was consumed and the circulating fans were not used, so that mixing was natural. The results of both experiments are plotted in Fig. 1, with the background levels of RSP subtracted. The data points generally represent 1-minute average values. The differences in mixing dramatically affect the slopes of the decay curves.

The theoretical curves shown in Fig. 1 were generated by fitting the data points from the decay curves to Eq. 3 with a regression analysis;  $A_{in}$  and  $\tau_e/\text{min}$  were determined and used to calculate the growth curves from Eq. 1, case (ii). The ratio of the slopes of the decay curves for ideal and natural mixing yields the mixing factor for the room. Table 2 gives the values obtained for the various parameters. The value of the mixing factor obtained is in good agreement with the expected value given in Table 1 for the case of infiltration and natural draft. The growth curve for the case of natural mixing (experiment 2) shows a poor fit initially because of the effect of the warm smoke rising to the ceiling and remaining

Table 3. Field survey of indoor RSP in the absence of smoking.

Locale	Room volume (m <sup>3</sup> )	Per-sons per room	Per-sons per 100 m <sup>3</sup>	Indoor RSP level* ( $\mu\text{g}/\text{m}^3$ )	Average time per RSP sample (min)	Out-door RSP level† ( $\mu\text{g}/\text{m}^3$ )	Comment
Crepe restaurant (Washington, D.C.)	124	43	35	29	20	44	No smoking section; aroma of frying crepes evident
Sandwich restaurant (Laurel, Md.)	326	40	12	53	21	40	No smoking section; near kitchen; three smokers in smoking section
Sandwich restaurant (Laurel, Md.)	326	55	17	51	21	55	No smoking section; near kitchen; one smoker in smoking section
Fast-food restaurant (Bowie, Md.)	1,400	22	1.6	38	7		Aroma of hamburgers frying
Private residence (Seabrook, Md.)	120	11	9	24	20		Cocktail party; one candle burning 6 m from RSP detector
Private residence (Bowie, Md.)	124	1	0.8	44	15		One hour after sweeping basement floor
Private residence (Greenbelt, Md.)	22	2	9	24	6		Natural mixing
Private residence (Greenbelt, Md.)	22	2	9	55	1		Two fans moving 110 m <sup>3</sup> of air per minute <sup>§</sup>
Private residence (Glenn Dale, Md.)	29	7	24	57	5		One fan moving 55 m <sup>3</sup> of air per minute <sup>§</sup>
Conference room (Greenbelt, Md.)	113	10	9	53	10		Two fans moving 50 m <sup>3</sup> of air per minute <sup>§</sup>
Public library meeting room (Bowie, Md.)	1,415	30	2.1	29	30		During piano recital
Library of Congress (Washington, D.C.)	27,000	130	0.48	30	10		Main reading room
Church (Bowie, Md.)	4,224	300	7	30	42		During Sunday service
Bagel bakery (Yonkers, N.Y.)	510	30	6	25	10	8	Aroma of baking bagels evident
Private residence (Hawthorne, N.Y.)	150	17	11	26	16		During dinner party

\*Mean  $\pm$  standard deviation for the Washington area samples,  $40 \pm 13 \mu\text{g}/\text{m}^3$ . <sup>†</sup>Duration of sampling, 3 minutes. <sup>‡</sup>Experiment 2 background. <sup>§</sup>Experiment 1 background. <sup>¶</sup>Experiment 3 background. <sup>||</sup>Experiment 4 background.

2023380246

out of the detector's range for about 2 minutes. Experiment 3 demonstrated that Eq. 3 is valid under more general conditions, that is, when a cigarette is actually smoked.

We conclude that these experiments show that for the unventilated room, Eq. 3, the reduced form of Eq. 1, is useful in describing the growth and decay of cigarette smoke particulates.

#### Ventilated Growth and Equilibrium of Tobacco Smoke

An experiment was conducted in a ventilated conference room of a modern office building to test Eq. 1 in the case of removal of uniformly generated tobacco smoke by both decay and ventilation. The experiment involved measuring the growth of cigarette-generated RSP from background levels to near equilibrium. Analysis of the RSP-versus-time curve determines  $\tau_v$ , the mean ventilation time, and  $C_T$ , the total RSP liberated from the combined sidestream and exhaled mainstream smoke.

The RSP detector was located in the geometric center of the 113-m<sup>3</sup> room. Two fans with antiparallel exhausts were used to establish a vigorous circulation of 100 m<sup>3</sup>/min. The ideal ventilation time  $\tau_v$ , calculated from the volumetric flow rates of the ventilation system, was 49.2 minutes for a complete change of air. Thirty-two cigarettes were smoked in 49

minutes by a relay of seven smokers, with an average of four persons smoking at any given time. When smoking their own brands, they averaged 9.8 minutes per cigarette; when smoking cigarettes supplied to them, they averaged 5.8 minutes per cigarette. All butts were collected and the amount of tobacco consumed was measured for each cigarette. The estimated mainstream TPM ( $M$ ) (tar plus nicotine) generated by the 32 cigarettes was determined by weighting the TPM values for each cigarette (24) by the fraction of tobacco consumed, and adding the results to obtain  $M = 418 \text{ mg}$  [TPM is emitted from cigarettes at a linear rate after the fourth puff (25)].

Figure 2 shows the growth against time of RSP from the cigarette smoke. The data points are corrected for background RSP levels and are 2-minute averages. A regression analysis using Eq. 1 yields  $A_{eq} = 1947 \mu\text{g}/\text{m}^3$  and  $\tau_v = 14 \text{ minutes}$ , with a coefficient of determination = .964 (from Eq. 2;  $\tau_v = 19.5 \text{ minutes}$ ). Finally,  $C_T$ , or the total amount of RSP liberated in the room during the entire smoking period, 772 mg, is calculated by using Eq. 1:  $C_T/M = 1.85$ . This ratio represents a weighted average for six different brands of filter cigarettes that together commanded a 23 percent share of the market in 1976 (26).

From the goodness of fit of the theory to the data and from the observation of predicted interactive behavior among mixing, growth, and decay processes for RSP from cigarette smoke, it appears that all the room-specific factors affecting the removal of tobacco smoke (ventilation, decay, and mixing) can be combined into a single time constant  $\tau_v$ , which can be determined for any room by regression analysis of the decay or growth-equilibrium curves, or by calculation from the equilibrium concentration if the smoke generation rate and room volume are known. The ratio of the slopes of the decay curves for ideal and natural mixing yields the mixing factor. We conclude that Eq. 1 is a useful tool for predicting the levels of tobacco smoke in both ventilated and unventilated occupied space.

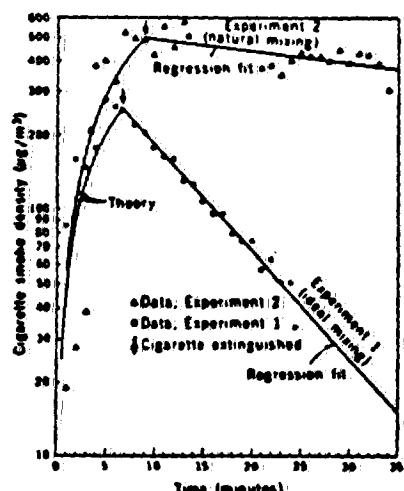


Fig. 1. Theoretical predictions versus experimental results for the growth and decay of RSP from a smoldering, average-tar cigarette in a 22-m<sup>3</sup> unventilated room. The dramatic difference in the slopes of the decay curves reflects the difference in room air turbulence (mixing) for the otherwise similar experiments.

times, effective ventilation rates, and the TPM values of various brands of cigarettes. The problem may be simplified by assuming that smoking is a random process when it occurs among large groups of people. It follows that cigarette smoke RSP values may be treated as equilibrium values; that all of the smokers may be treated as habitual smokers who smoke identical average-tar cigarettes in the same way at the same average rate, uniformly distributed over a 16-hour day. An average smoking rate  $r$  of two cigarettes per hour is calculated from the 1975 figures for the number of U.S. smokers and the U.S. domestic cigarette consumption (8). In 1978, the sales-weighted average mainstream TPM value  $M$ , was 17.6 mg for all the cigarettes sold in the United States (7). The estimated emission rate  $C_0$  (combined sidestream plus exhaled mainstream TPM) from a habitual smoker is given by  $E = 1.85 r M_0 = 65 \text{ mg/hour}$ , where 1.85, used for the ratio  $C_0/M_0$ , is taken from the conference room experiment. Physically observable in any field survey of smoking is  $n_a$ , the number of burning cigarettes (the number of "active" smokers);  $n_a$  can be related to the number of habitual smokers  $n_m$  by considering that the average time for smoking a cigarette is 10 minutes (2, 6). This number and the previously calculated average smoking rate indicate that  $n_m = 3 n_a$ .

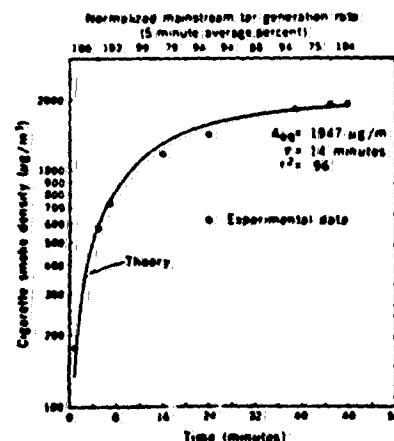


Fig. 2. Theoretical predictions versus experimental results for the attainment of equilibrium  $A_{eq}$  for the combined emission of sidestream and exhaled mainstream cigarette smoke from four chain-smokers in a 113-m<sup>3</sup> conference room with well-mixed ( $m = 1$ ) ventilation in a modern office building. Under natural mixing conditions, about 11 habitual smokers would generate an equivalent equilibrated concentration of smoke. This many smokers would be expected in a group of 33 adults (well within the capacity of this 50-person conference room).

2023380247

From the equilibrated form of Eq. 1, we determine that

$$R = A_{eq} = 650 \frac{D_s}{C_a} \quad (4)$$

where  $R$  is the smoker-generated equilibrium RSP level in micrograms per cubic meter,  $D_s$  is the density of active smokers (number per  $100\text{ m}^3$ ), and  $C_a$  is the effective rate for the removal of cigarette aerosol (air changes per hour), with  $C_a = 60/r$ .

The aerosol sampling described in this article was performed from late March through early June 1978 in the Washington, D.C., metropolitan area. The MMD (seasonal average) of the outdoor urban aerosol for Washington in 1970 was  $0.5\text{ }\mu\text{m}$ , with 90 percent of the aerosol mass less than  $3\text{ }\mu\text{m}$  in aerodynamic diameter and lognormally distributed (27).

It is important to note that all of the RSP measurements we report represent time-averaged values.

Factors other than tobacco smoke may contribute to indoor RSP. These include infiltration of outdoor RSP, cooking, dust raised by indoor traffic, and industry. By restricting the sampling to nonindustrial indoor locations where tobacco smoking is absent, the effect of the remaining variables may be assessed. Table 3 gives the RSP levels for several indoor spaces in which smoking did not take place: three restaurants, four private residences, an office building conference room, two libraries, and a church during services. The mean of these measurements is  $40\text{ }\mu\text{g}/\text{m}^3$ . In three instances, fans were mixing the air at a high rate and RSP levels were elevated, apparently because of dust entrainment. No correlation between the volumetric density of people (occupancy) and RSP is evident. Hemperly (28), in sampling RSP in Houston, found similar RSP levels in two schools, a library, and a museum—all nonsmoking areas.

Table 4 gives the results of RSP sampling in nonsmokers' automobiles traveling along two major commuter highways (Route 50 in Maryland and U.S. 1-295 in Washington) during the rush hour. The samples were taken on different days and were measured in different vehicles. In all cases, the windows were slightly open and the ventilation fans were running. The mean of the data,  $38\text{ }\mu\text{g}/\text{m}^3$ , is not very different from the mean of the indoor readings,  $40\text{ }\mu\text{g}/\text{m}^3$  (Table 3).

The impact of actual ventilation practice on ambient RSP levels from smoking was investigated at eight restaurants, three cocktail lounges, two bingo games, a dinner-dance, a bowling alley, a sports

Table 4. Levels of RSP in nonsmokers' cars during rush-hour traffic on a busy commuter highway in Washington, D.C., measured with the vehicles' windows slightly open and the ventilation fans running. Each car carried four persons and had a volume of  $2\text{ m}^3$ , so that the occupancy was equal to 200 persons per  $100\text{ m}^3$ .

Date	Time	Sampling time (min)	RSP level ( $\mu\text{g}/\text{m}^3$ )
23 March	a.m.	10	40
23 March	p.m.	35	20
24 March	a.m.	20	54
28 March	a.m.	26	49
31 March	a.m.	8	25
Mean $\pm$ standard error			$38 \pm 15$

arena, and a hospital emergency waiting room. For contrast, one unventilated private residence was sampled during a cocktail party. With the exception of the hospital waiting room and the hotel bar, each space sampled represented the major part of the building and was subject to ventilation requirements specified by building codes. Sampling was generally performed well after opening time to ensure that an approximately steady-state level of smoking had been reached.

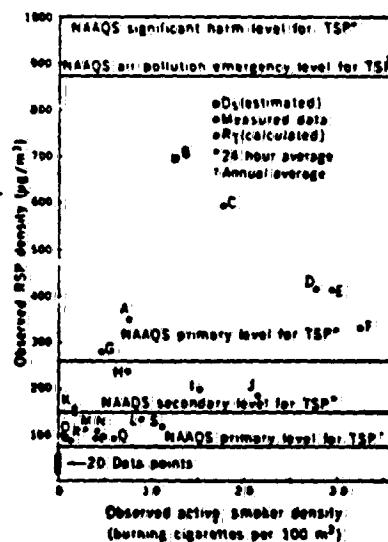


Fig. 3. Results of a field survey of short-term time-averaged levels of RSP in 38 enclosed spaces (see Tables 3, 4 and 5). Levels corresponding to federal standards for TSP are indicated for comparison only. The microenvironments include ten restaurants, three cocktail lounges, two bingo games, a dinner-dance hall, a bowling alley, a sports arena, two libraries, a church, a hospital waiting room, five vehicles, and five residences. The letters A through S correspond to those given in Table 5. The effective air change rates for microenvironments A and S are known from experiments to be  $C_a = 1.3$  and  $9.2$ , respectively.

The piezobalance and a stopwatch were used to take tabletop-level RSP samples for periods ranging from 10 to 50 minutes (mean, 20 minutes). The piezobalance was equilibrated in advance to avoid errors due to changes in temperature or humidity.

The room dimensions were estimated and the number of active smokers was sampled periodically throughout the measurement period and averaged. It was usually not possible to sample the premises when there was no smoking; in most cases, the RSP outside the premises was sampled for comparison. Table 5 gives the results of the measurements. Figure 3 shows the average density of active smokers (defined as the number of burning cigarettes per  $100\text{ m}^3$ ) plotted against the total indoor RSP sampled. As a guide to whether a given datum is "high" or "low," the National Ambient Air Quality Standards (NAAQS) for total suspended particulates (TSP) are also shown. Since a specific averaging time is incorporated into these standards, violation of the standard is not demonstrated here. However, repeated exposure to such elevated levels can lead to "violation" of the annual standards, as will be shown later. Note that all the data for finite smoker density exceeded the level of the annual primary (health-related) NAAQS, whereas none of the data for zero smoker density exceeded this level. Further, the background RSP measured outside the smoking premises suggests that the source of these elevated levels was not the outdoor air. The mean and the standard deviation for the outdoor RSP are  $46 \pm 13\text{ }\mu\text{g}/\text{m}^3$ , and in every case the outdoor level is less than the indoor. In certain cases, indoor controls are available. In bingo game 2, held in the nave of a church, the active smoker density was  $0.47$  per  $100\text{ m}^3$ , the occupancy was  $3.6$  persons per  $100\text{ m}^3$ , and the RSP density was  $279\text{ }\mu\text{g}/\text{m}^3$  (Table 5). By contrast, during the tobacco smoke-free religious services, despite an occupancy of  $7.0$  persons per  $100\text{ m}^3$ , 30 burning votive candles, and several processions, the RSP density was  $30\text{ }\mu\text{g}/\text{m}^3$ . The elevated RSP levels in the bingo game clearly appear to be due to smoking. Similarly, measurements taken in the nonsmoking section of a sandwich restaurant showed considerably lower levels than in the smoking section, indicating that the contribution of smoking to RSP was much larger than the effect of cooking, even at the low cigarette densities shown (Table 5). Figure 3 shows that, in general, RSP levels increase with active smoker density, although there is

2023380248

considerable scatter in the data. The question now is whether Eq. 1 is useful in explaining this scatter.

We hypothesize that the levels of RSP for finite  $D_s$  (Fig. 3) are due to near-equilibrium levels of cigarette smoke adding to much smaller background levels, and that the scatter in the RSP levels for fixed cigarette density is due primarily to differences in the mean ventilation time  $\tau$ . Analyzing the background corrected data given in Table 5, we use Eq. 4 to calculate a range for  $C_{st}$  between 1.2 and 10.7 air changes per hour;  $C_{st}$  is used instead of  $\tau$  to facilitate comparison with building code-specified ventilation rates. The range determined for  $C_{st}$  is consistent with two known values of  $C_{st}$ , derived from the cocktail party and roadside restaurant experiments (Table 5).

The  $C_{st}$  for tobacco aerosol is affected by the rate of mechanical ventilation and infiltration, the rate of smoke adsorption, and mixing. The range of mechanical ventilation and infiltration can be calculated from tables of standards deter-

mined by the American Society of Heating, Refrigerating, and Air Conditioning Engineers (ASHRAE) (29), the authority specified by the local building code (30). For each premise listed in Table 5, the recommended maximum number of outdoor air changes per hour (based on the estimated floor area, maximum occupancy, and volume) was calculated from the ASHRAE tables; a two-thirds recirculation of air (the maximum permitted by ASHRAE) was assumed. This yielded a range of 0.7 to 9.4 air changes per hour. Infiltration, resulting mainly from the opening of doors, was estimated from the actual occupancy during the sampling (29); we assumed a 100 percent turnover of occupants per hour. This was added to the calculated mechanical ventilation rates, giving a final estimated range of  $1.3 \leq C_{st} \leq 13.4$  air changes per hour, where  $C_{st} = 60/\tau$ .

The practical range of physical decay from adsorption for cigarette aerosol can be computed from our experiments and the literature. Most establishments pos-

sess simple filters that are relatively ineffective at removing tobacco smoke (22). The shortest ideal decay time measured (in experiment 1) was equivalent to six air changes per hour (Table 2). By contrast, Penkala and DeOliviera (15) measured a mean life for tobacco smoke, under uniform mixing in a chamber with unreactive walls, equivalent to one air change per hour. These two extremes give an estimated range of  $1 \leq C_{eq} \leq 6$  air changes per hour for RSP from tobacco smoke, where  $C_{eq} = 60/\tau_e$ .

The range of mixing  $m$  appropriate for the spaces listed in Table 5 is  $1/4 \leq m \leq 1/2$ , as determined from Table 1. By using Eq. 2, a theoretical range of mean air change rates,  $1/2 \leq C_{sth} \leq 10$  air changes per hour, is calculated from the estimated ranges for  $C_{st}$ ,  $C_{eq}$ , and  $m$ . This is consistent with the 1 to 11 air changes per hour determined with our model from the experimental results. In other words, the variations in the observed RSP density for fixed cigarette density can be phenomenologically ac-

Table 5. Field survey of indoor RSP sampled in the presence of smoking. Where the standard deviation is given, the value is an average of 2-minute samples; where it is not given, the sampling time is the averaging time.

Locale	Estimated volume (m <sup>3</sup> )	Average number of smokers	Indoor sampling time (min)	Average occupancy (persons)	Active smoker density per 100 m <sup>3</sup>	Indoor RSP (μg/m <sup>3</sup> )	Outdoor RSP (μg/m <sup>3</sup> )	Outdoor sampling time (min)	Occupants smoking (%)	Date	Time
A. Cocktail party*	268	2	15	14	0.75	351 ± 38		14	8 April	9:00 p.m.	
B. Lodge hall	3,168	40†	50	350	1.26	697 ± 28	60	6	11‡	31 March	11:00 p.m.
C. Bar and grill	507	9	18	75	1.78	589 ± 28	63	6	12	21 March	8:00 p.m.
D. Firehouse bingo	541	10.5	16	125	2.77	417 ± 63	51	15	8.4	27 March	10:00 p.m.
E. Pizzeria	170	5	32	50	2.94	414 ± 58	40	5	10	14 April	8:00 p.m.
F. Bar/cocktail lounge	216	7	26	55	3.24	334 ± 120	50	5	13	25 March	10:00 p.m.
G. Church											
Bingo game	4,224	20	8	150	0.47	279 ± 18			13	31 March	10:00 p.m.
Sunday service	4,224	0	31	300	0	30			0	13 May	11:00 a.m.
H. Inn	338	2.5	12	70	0.74	239 ± 9	22	10	3.5	23 March	1:00 p.m.
I. Bowling alley	918	14	20	128	1.53	202 ± 19	49	5	11	25 March	8:00 p.m.
J. Hospital waiting room	93	2	12	19	2.15	187 ± 52	58	6	11	28 March	10:30 p.m.
K. Shopping plaza restaurant:											
Sample 1	1,369	2.5	18	95	0.18	153 ± 8	59	5	2.6	24 March	7:30 p.m.
Sample 2	1,369	2.5	18	50	0.18	163 ± 4	36	10	5	24 March	9:30 p.m.
L. Barbeque restaurant	225	2	10	25	0.89	136 ± 17			8	24 March	9:00 p.m.
M. Sandwich restaurant A:											
Smoking section	781	2.25	20	30	0.29	110 ± 36	40	5	7.5	25 March	8:00 p.m.
Nonsmoking section	326	0	20	40	0	55 ± 5	40	5	0	25 March	7:30 p.m.
N. Fast-food restaurant											
Sample 1	360	1.5	40	30	0.42	109 ± 38			5	26 March	2:00 p.m.
Sample 2	360	0	7	30	0	30			0	26 March	1:30 p.m.
O. Sports arena	823,000	739†	12	6,700‡	0.09	94 ± 13	24	5	11‡	29 March	10:00 p.m.
P. Neighborhood restaurant/bar	250	1	12	35	0.40	93 ± 17			2.9	25 March	8:30 p.m.
Q. Hotel bar	169	1	12	25	0.59	93 ± 2			8		2:30 p.m.
R. Sandwich restaurant B:											
Smoking section	781	1	8	30	0.13	86 ± 7	55	5	3.3	14 April	11:00 a.m.
Nonsmoking section	326	0	21	50	0	51	35	5	0	14 April	1:30 p.m.
S. Roadside restaurant											
Sample 1	90	1	18	5	1.12	107§		20	29 March	3:00 p.m.	
Sample 2	90	0	2	3	0	30		0	29 March	3:00 p.m.	

\*Only the cocktail party microenvironment was unventilated.

†Estimated. See U/I.

‡Paid attendance.

§Calculated equilibrium value.

2023380249

counted for by ventilation, recirculation, infiltration, decay, mixing, and average smoking behavior. We conclude that the finite  $D$ , RSP levels shown in Fig. 3 are indeed generated primarily by cigarette smoke and that this is consistent with the predictions of our model.

#### The Range of Public Exposure

We can now model the full range of exposure of the nonsmoking public to cigarette smoke. Equation 4 may be rewritten as:

$$R = 25.6 \frac{P_a}{C_s} \quad (5)$$

where  $P_a$  is the occupancy (persons per 1000 square feet). (The volumetric measure is implicit, assuming a 10-foot ceiling.) The  $P_a$  is three times the density of habitual smokers  $D_{hs}$  and nine times the density of active smokers  $D_a$  (31). A family of RSP curves is generated from Eq. 5 by varying  $C_s$  and  $P_a$  over their ranges. Representative samples of this family are plotted in Fig. 4. A lower limit for  $C_s$  of about one-half to one mean air change per hour has been determined experimentally and theoretically for removal of cigarette aerosol from private dwellings ventilated by infiltration and from commercial establishments whose mechanical ventilation is poor. A realistic upper bound for  $C_s$  may be obtained from the well-ventilated environment of the commercial airliner. A mechanical (design) ventilation rate of 15 to 20 air changes per hour with no recirculation is typical of the Boeing 707 (32). The best ideal decay rate measured in the experiments was six air changes per hour. Assuming a mixing factor of unity, we calculate an upper limit for  $C_s$  of 26 air changes per hour. The practical range for  $P_a$  is obtained from the ASHRAE (29), which specifies mechanical ventilation rates for typical average occupancies in various structures. For commercial structures, these densities (in persons per 1000 square feet) range from 10 for general office space to 70 for dining rooms to 150 for such places as stand-up bars, auditoriums, arenas, and commercial aircraft. The design ventilation rate  $C_v$  is typically determined from both the design occupancy and the intended use of the structure. For example, 15 to 25 cubic feet per minute per occupant is specified for general office space, 10 to 20 for dining rooms, and 30 to 40 for cocktail lounges. In 1975, ASHRAE Standard 90-75, "Energy conservation in new building design," decreased these rates by factors

of one-half to one-third. ASHRAE Standard 62-73 is currently being revised to specify higher rates of ventilation for premises in which smoking is permitted. How effective would increases in  $C_v$  be in lowering the levels of RSP from tobacco smoke? Equation 5 shows that such levels decrease only exponentially with increasing  $C_v$ . Furthermore, as Kallika *et al.* (33) observed, "the current practice of recirculation or reuse of air is largely dictated by the economics of heating and cooling, with little regard for changes in indoor air quality." That is, ventilation may be subject to arbitrary reduction by building management or by legislative or bureaucratic fiat; in many nonurbanized areas, it may not even be regulated by building codes (34).

Figure 4 illustrates the estimated range

of exposure of the nonsmoking public to RSP from cigarette smoke. The actual dose of RSP is clearly a function of personal activity patterns; differences in respiration rate also affect the dose. Many different scenarios can be imagined. In the following, we express a range of RSP burdens from the cigarette aerosol relative to a typical RSP ambient background level. For an air shed (air quality control region) that is in compliance with the annual secondary (public welfare) NAAQS for TSP of 60  $\mu\text{g}/\text{m}^3$ , the RSP fraction of the ambient aerosol is conservatively estimated at 50  $\mu\text{g}/\text{m}^3$  and is likely to be composed largely of combustion-produced sulfates (35). Since the particle size distributions of this fraction and the cigarette aerosol are both in the respirable range, we first compare them on a mass basis, without regard for differences in the chemical composition of each.

Let A, B, C, and D be nonsmokers who dwell in the same air shed and who breathe at the average rate of 20  $\text{m}^3/\text{day}$ . All have different occupations and lifestyles that lead, as we shall see, to dramatically different RSP burdens.

Nonsmoker A is a mailman who walks a regular route and is able to live in a completely tobacco smoke-free environment. He is exposed only to the background ambient and therefore inhales 365 mg of RSP annually.

Nonsmoker B is an office worker who works a 40-hour week 50 weeks per year in a 40- $\text{m}^3$  office with two other persons, one of whom is a habitual smoker. Replacing  $D_h$  in Eq. 4 with  $D_h/3$ , we find that B's mass RSP exposure is more than three times that of A (we calculate an expected  $C_s$  of 1.1 for office buildings).

Nonsmoker C is a musician who entertains in a popular, poorly ventilated nightclub 8 hours nightly, 5 nights per week, 50 weeks per year. The average  $P_a$  in the club is 100 persons per 1000 square feet (about 33 smokers). Further, C shares a 100- $\text{m}^3$  apartment with a roommate who is a chain smoker. C is exposed to the roommate's smoke 5 hours per day, 7 days per week, annually. By using Eqs. 4 and 5 and a  $C_v$  of one air change per hour, we find that C's mass RSP burden is more than 15 times that of A.

An alternative way of approaching the excess RSP exposure is in terms of cigarette equivalents. The cigarette with the least tar in the May 1978 FTC scale has 0.55 mg of TPM. In these terms, B's excess RSP burden is equivalent to 5 cigarettes per day and C's burden to 27 cigarettes per day. However, this may un-

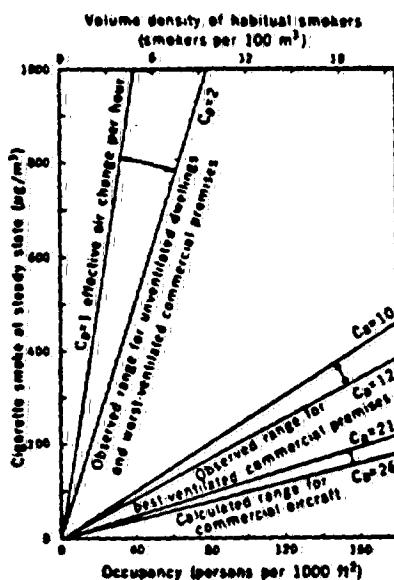


Fig. 4. Theoretical steady-state density of respirable particulates from environmental cigarette smoke in habitable indoor spaces, as related to the design occupancy  $P_a$ . On the average, one-third of adults are habitual smokers; for every three such smokers, we calculate that an average of one cigarette burns constantly throughout a 16-hour day. According to standard engineering criteria (29), occupancy and the type of microenvironment determine the design rate of mechanical ventilation  $C_v$ . The effective air change rate ( $C_e$ ) for the removal of tobacco aerosol from room interiors is determined by  $C_v$ , by mixing, and by the rate of adsorption of tobacco particles on room surfaces. Generally  $C_v$  and hence  $C_e$  increase with  $P_a$ . [Typical  $P_a$  (in persons per 1000 square feet) ranges from 10 for office buildings to 70 for restaurants to 150 for bars, sports arenas, and aircraft (29, 32).] We estimate the practical range of  $C_e$  to be from 1 to 12 air changes per hour. It appears that over the combined practical ranges of  $P_a$  and  $C_v$ , repeated exposure to tobacco smoke can lead to annual RSP burdens that violate the primary annual NAAQS.

2023380250

derestimate the true impact, since nonsmokers have greater sensitivity to smoke than smokers (7).

Nonsmoker D is a flight attendant who spends 40 hours per week, 50 weeks per year on board a commercial airliner with a  $C_6$  of 23 air changes per hour. The average  $P_a$  on the plane is 150 persons per 1000 square feet. D's RSP burden is nearly twice that of A. Even with one of the best ventilation systems in use, the high density of smokers causes a substantial increase in mass RSP inhaled by D.

The following three considerations may help to place these scenarios into perspective. First, an annual exposure 1.5 times that of A is sufficient to exceed the primary annual NAAQS; the exposure of D, B, and C to RSP all violate the standard by factors of 1.2, 2, and 10, respectively. Second, pulmonary clearance studies show that the half-life of inert respirable particles (2.8  $\mu\text{m}$  in MMD) in the lungs of nonsmokers is ~ 70 days (36); residence of RSP in the lungs is prolonged. Third, in a series of pulmonary lavage studies on 400 nonrandomly selected volunteers (250 nonsmokers and 150 smokers) (37), two of the nonsmokers had tarry lavage fluids with pigmented pulmonary alveolar macrophages strikingly similar to those found in smokers. In these two volunteers, the levels of aryl hydrocarbon hydroxylase, an inducible carcinogen-detoxifying pulmonary enzyme, were intermediate in value between the levels found in smokers and most nonsmokers. These findings were attributed to the effects of exposure to tobacco smoke (38).

#### Health Policy Implications

There is good toxicologic evidence that elevated levels of particulates in outdoor air, perhaps in combination with other pollutants, cause illness and death during air pollution episodes (particulate levels in excess of 1000  $\mu\text{g}/\text{m}^3$  per 24 hours). There is much epidemiologic evidence, some of it conflicting, that lower levels of particulates, perhaps in combination with other pollutants, affect respiratory health adversely when exposure to them is sustained (39). (This evidence has been used to establish the thresholds for harm on which the primary annual NAAQS for TSP is based.) There is excellent toxicologic evidence that mainstream cigarette smoke causes chronic obstructive pulmonary disease (7, 40). Epidemiological evidence, some of it conflicting, indicates that exposure to to-

bacco smoke in the home affects respiratory health adversely (7, 41). Finally, there is excellent evidence that mainstream cigarette smoke causes cancer in many organs (7). Sidestream smoke is chemically identical to mainstream smoke, and typically is more concentrated (2). Coke-oven emissions, which chemically are similar to tobacco smoke, are associated with increased rates of many forms of cancer in coke-oven workers (42). Animal studies demonstrate that the particulate phase of tobacco smoke contains numerous potent carcinogens and tumor promoters, initiators, and accelerators (7). One of these, benzo[a]pyrene, was detected at a concentration of 40 parts per million in ambient tobacco smoke (13). Strong evidence supports a correlation between the magnitude of long-term exposure to carcinogens and the incidence of cancer (43). Therefore, given the efforts by public health authorities to eliminate involuntary public exposure to saccharin and the fire retardant Tris—which have, respectively, one fifty-thousandth and one-tenth the experimental carcinogenic potency of benzo[a]pyrene alone (44, 45)—similar efforts to prevent involuntary exposure to ambient tobacco smoke (46) appear justified.

#### Conclusions

We have defined the probable range of exposure of the nonsmoking public to a common pathological aerosol, cigarette smoke. We showed, both experimentally and theoretically, that under the practical range of ventilation conditions and building occupation densities, the RSP levels generated by smokers overwhelm the effects of ventilation and inflict significant air pollution burdens on the public. Our observations show that levels of RSP in places where tobacco is smoked greatly exceed levels found in smoke-free environments, outdoors, and vehicles on busy commuter highways. Our experimental results are consistent with the large differences in 24-hour average RSP levels reported for smoking and nonsmoking homes in the Harvard Six-City Study (47), with a survey of short-term RSP levels in commercial and public buildings in Houston (28), and with other studies of tobacco-generated TSP (7, 11-13).

Attempts to reduce RSP levels from smoking by increasing the rate of mechanical ventilation or the efficiency of filtration yield exponentially diminishing returns for linear increases in ventilation

(and cost). Moreover, efforts to conserve energy in buildings will decrease ventilation rates (48). Therefore, increased ventilation does not appear to be a solution to the problem. Indoor air is a resource whose quality should be maintained at a high level. Smoking indoors may be incompatible with this goal (33, 49).

Further research is necessary to define the integrated particulate exposure of various segments of the population; compliance with the NAAQS, as indicated by the establishment of outdoor TSP sampling stations, does not imply protection of the public from excessive RSP burdens. Repeated exposure to ambient cigarette smoke imposes air pollution burdens on nonsmokers that exceed the primary annual NAAQS. It appears that the RSP burdens from ambient tobacco smoke are so large that they must be incorporated explicitly in future epidemiological assessments (30, 31) of the relation between particulate levels and morbidity or mortality.

The Clean Air Act of 1970 and its amendments mandate the control of public exposure to outdoor TSP. However, little legislative attention has been devoted to the quality of indoor air—other than the passage of the Public Health Service Act of 1978, which provides for an ongoing study of the health costs of indoor air pollution. Clearly, indoor air pollution from tobacco smoke presents a serious risk to the health of nonsmokers. Since this risk is involuntary, it deserves as much attention as outdoor air pollution.

*Note added in proof:* A very recent epidemiological study concluded that long-term exposure to tobacco smoke, limited to the work environment only, is deleterious to the nonsmoker and significantly reduces small-airway function to the same extent as smoking one to ten cigarettes per day. This is consistent with scenario B (52). ASHRAE Standard 62-73R, a proposed standard for ventilation required for minimum acceptable indoor air quality, has been published (see 29). Using data supplied in the standard, we calculate a  $C_6$  of  $\leq 1.28$  for office buildings where smoking is permitted.

#### References and Notes

1. W. R. On, "Human activity patterns: A review of the literature for air pollution exposure estimation," (Environmental Protection Agency, Washington, D.C.), in press.
2. U. R. Hong, *Eur. Health Perspect.* 2, 117 (1972).
3. B. G. Ferris, Jr., *J. Air Pollut. Control Assoc.* 28, 482 (1978); R. E. Binder, C. A. Mitchell, H. R. Hoskin, A. Bouhuys, *Arch. Environ. Health* 31, 277 (1976).
4. T. D. Surling and D. M. Kobayashi, *Environ. Res.* 13, 1 (1977).

2023380251

3. C. R. Thompson, E. G. Hensel, G. Kaus, *J. Pollut. Control Assoc.* 23, 88 (1973).
4. D. P. Bridge and M. Corn, *Environ. Res.* 8, 192 (1972).
5. *Smoking and Health: A Report of the Surgeon General* (Publ. 78-50066, Department of Health, Education, and Welfare, Washington, D.C., 1979).
6. D. Horn, paper presented to the Commission on Smoking Policy of the American Cancer Society, Los Angeles, 22 March 1977; "The Tobacco Situation" (Publ. T3-166, Department of Agriculture, Washington, D.C., 1978).
7. F. Speer, *Arch. Environ. Health* 16, 643 (1968); H. Savel, *ibid.* 21, 146 (1970); G. Greene, *J. Am. Med. Assoc.* 239, 2125 (1978); C. B. Barad, *Occup. Health Saf.* 48 (No. 1), 21 (1979).
8. M. Russell and C. Feyerabend, *Lancet* 1978-I, 179 (1978).
9. M. Corn, *Scand. J. Respir. Dis. Suppl.* 91, 21 (1974).
10. J. E. Cuddeback, J. R. Donovan, W. R. Burg, *J. Am. Ind. Hyg. Assoc.* 37, 263 (1976).
11. L. P. Elliot and D. R. Rowe, *J. Air Pollut. Control Assoc.* 28, 635 (1975).
12. A. Turb, *J. Am. Soc. Heat. Refrig. Air Cond.* 26, 55 (1963); in *Basic Principles of Sensory Evaluation* (American Society for Testing Materials, Philadelphia, 1968).
13. S. J. Penkala and G. DeOliviera, *Environ. Res.* 9, 99 (1975).
14. R. S. Brief, *Air Eng.* 2, 39 (1960).
15. G. J. Sem, *Methods and Standards for Environmental Measurement* (Spec. Publ. 464, National Bureau of Standards, Washington, D.C.), p. 191.
16. G. J. Sem and K. Tsurubayashi, *J. Am. Ind. Hyg. Assoc.* 36, 791 (1975).
17. ———, K. Homma, *ibid.* 36, 580 (1975).
18. M. Corn and N. P. Esman, *Am. Lab.* (July 1978), p. 19.
19. W. C. Hinds, *J. Am. Ind. Hyg. Assoc.* 39, 48 (1978); J. Portendorfer and A. Schraub, *Siedl.-Reinhalt. Luft* 32, 33 (1972).
20. P. E. McNeil, *Arch. Environ. Health* 30, 552 (1975).
21. Department of Health, Education, and Welfare, *Adult Use of Tobacco, 1975* (Center for Disease Control, Atlanta, 1976).
22. *Tar and Nicotine Content of Cigarettes* (Publ. 78-5703, Department of Health, Education, and Welfare, Washington, D.C., 1977).
23. C. H. Kieth and K. Teab, *Job. Sci.* 9, 65 (1965).
24. J. C. Maxwell, "Report on the tobacco industry," *Barrons*, 19 October 1976.
25. R. E. Lee, Jr., *Science* 178, 567 (1972).
26. W. Hemperly, thesis, University of Texas, Houston (1976).
27. *ASHRAE Standard 62-73 for Natural and Mechanical Ventilation* (American Society of Heating, Refrigerating, and Air Conditioning Engineers, New York, 1973).
28. *BOCA Basic Building Code/1973* (Building Officials and Code Administrators, Chicago, ed. 6, 1973).
29. Since about one-third of U.S. adults are smokers (23), in principle, one-ninth (11.1 percent) of every group of average adults will be smoking at any one time under most social conditions. The larger the group considered, the closer the actual number should be to 11.1 percent. Table 5 shows that, averaged over all data points for which the number of active smokers was five or more, this percentage was  $11.2 \pm 1.8$ . Averaged over all data points, the percentage was  $8.8 \pm 4.6$ .
30. C. Robbins, private communication.
31. R. W. K. Kalita et al., *J. Am. Soc. Heat. Refrig. Air Cond. Eng.* 11, 44 (1970).
32. J. E. Woods, paper presented at the Human Factors Society Annual Meeting, Detroit, 19 October 1978; T. Pannikar, *Heat/Piping/Air Cond.* (December 1976), p. 23; Prof. Eng., Washington, D.C. (December 1976), p. 28; J. Botkin, private communication.
33. T. Pace and E. Meyer, Jr., paper presented at the Annual Meeting of the Air Pollution Control Association, Cincinnati, 24-29 June 1979.
34. D. Cohen, S. F. Arni, J. D. Brann, *Science* 204, 514 (1979).
35. R. R. Martin and G. A. Warr, *Hosp. Pract.* (September 1977), p. 97; T. L. McLamore et al., *Cancer Res.* 37, 1175 (1977).
36. R. R. Martin, private communication.
37. Airborne Particles (National Academy of Sciences-National Research Council, Washington, D.C., 1977).
38. A. Janos, H. Carp, D. K. Lee, R. T. Drew, *Science* 206, 1313 (1979); J. E. Gadsen, G. A. Fells, R. G. Crystal, *ibid.*, p. 1313.
39. W. Winkelstein and S. Kastor, *Arch. Environ. Health* 18, 544 (1969); *ibid.* 14, 162 (1967); *ibid.* 16, 401 (1968); *Am. J. Public Health* 59, 1134 (1969).
40. J. R. White and H. F. Freeb, *N. Engl. J. Med.* 303, 720 (1980).
41. We are grateful to the staff of the Prince George's County Health Department. In particular, we thank J. Koomz, J. H. O'Hara, and C. Gouin for their generous assistance with these experiments; W. Ott, J. Botkin, and H. Pillsbury for helpful discussions; and Thermosystems, Inc., and Gans & Pugh Associates for their generous loan of the piezobalance.

## Materials Science

On 23 May *Science* will publish an issue containing 20 articles devoted to Advanced Technology Materials. The issue will provide a sample of some of the more significant work being conducted in the major industrial research laboratories. The manuscripts have been prepared by leading industrial scientists who have delivered texts that are not only authoritative but also readable and interesting. Upper-division undergraduates, graduate students, and mature scientists will find the issue a valuable sample of applications of fundamental knowledge.

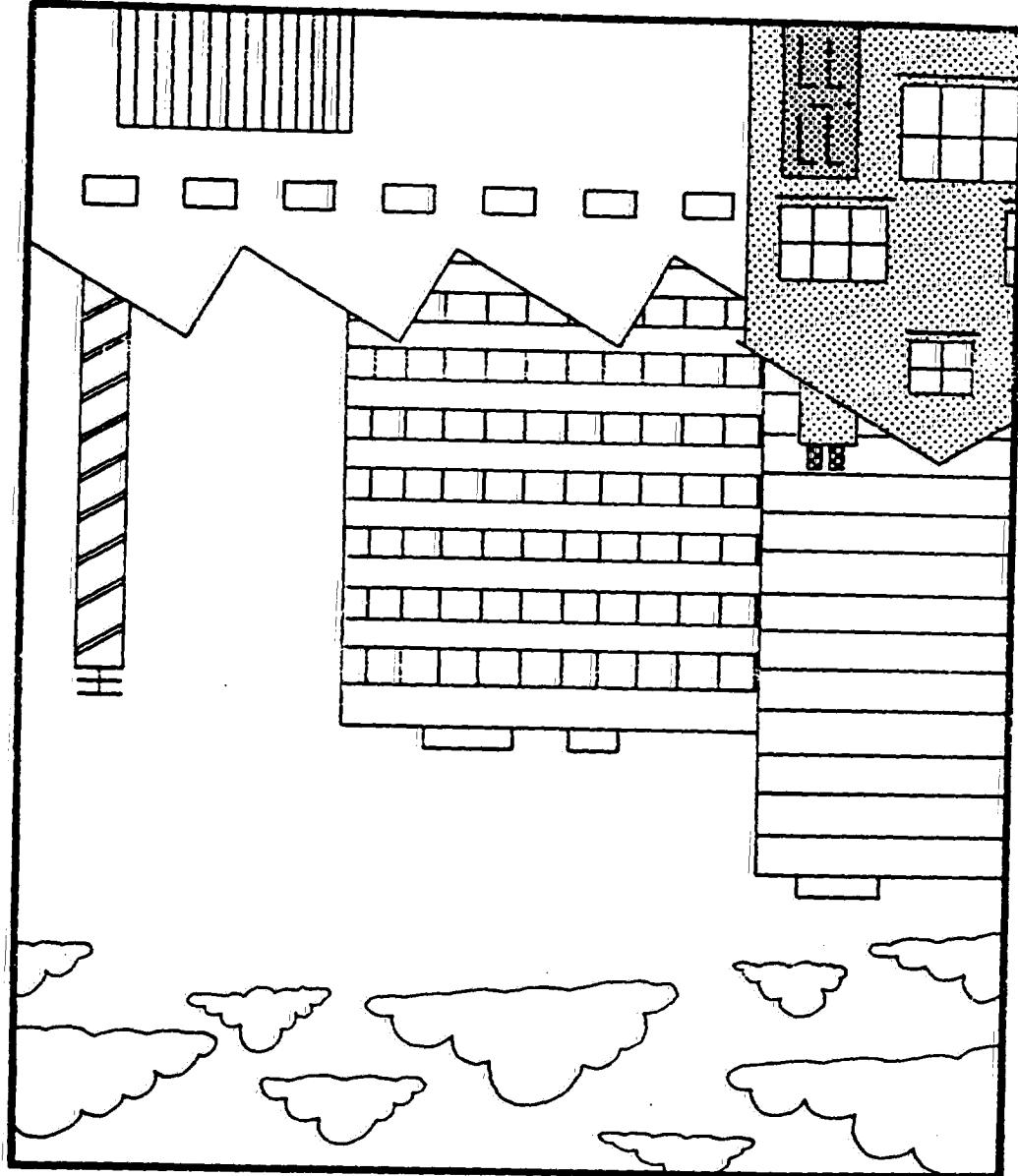
The topics covered include: New Polymers; Conductive Polymers; Multipolymer Systems; Fiber Reinforced Composite Materials; Heterogeneous Catalysts; Glassy Metals; High Strength Low Alloy Steels; Superconductors for High Current, High Fields; New Magnetic Alloys; High Temperature Ceramics; Gas Turbine Materials and Processes; Diamond Technology; New 3-S Compounds and Alloys; Molecular Beam Epitaxy; New Methods of Processing Semiconductor Wafers; Materials in Relation to Display Technology; Photovoltaic Materials; Magnetic Bubble Materials; Josephson Device Materials; and Biomedical Materials.

2023380252



Edited by R. Perry  
and P.W. Krik

2023380253



INDOOR AND  
AMBIENT AIR  
QUALITY

# **INDOOR AND AMBIENT AIR QUALITY**

**Sold and distributed by Publications Division,  
Selper Ltd., 79 Rusthall Avenue,  
Chiswick, London W4 1BN**

**ISBN 0 948411 04 X**



**Copyright held by  
©Selper Ltd., 1988**

**Published by  
Publications Division, Selper Ltd., London  
Printed by Printext Ltd., London**

**2023380254**

## RESULTS FROM SURVEY OF ENVIRONMENTAL TOBACCO SMOKE IN OFFICES IN OTTAWA, ONTARIO

John R. Carson\*, and Carol A. Erikson  
International Technology Corporation, 312 Directors Drive,  
Knoxville, Tennessee 37923, U.S.A.

(Received 11 May 1988; accepted 25 May 1988)

### ABSTRACT

A survey was performed in offices in Ottawa, Ontario to estimate non-smoker exposure to environmental tobacco smoke (ETS). Portable air sampling systems were used to sample 31 offices for nicotine, ultraviolet particulate matter (UV-PM, an ETS indicator), and carbon monoxide (CO). Mean concentrations of nicotine and UV-PM were 7.2, and 24  $\mu\text{g} \cdot \text{m}^{-3}$ , respectively. Exposure estimated from mean nicotine and UV-PM results were 0.004 and 0.001 cigarette equivalent per hour, respectively.

### INTRODUCTION

Two recently published reports (1,2) have concluded as a matter of public health policy that exposure to environmental tobacco smoke (ETS) is a health risk to non-smoking adults. These conclusions draw from epidemiological studies which attempt to determine the association between spousal smoking at home and disease incidence. These studies rely primarily on respondent questionnaires to classify ETS exposure. Few data are available regarding measured ETS exposures either in or out of the home. Because quantification of ETS exposure by questionnaire is imprecise, direct measurements of ETS are necessary. This report presents results from a survey performed in Ottawa from 25 to 28 August 1987 where ETS exposure levels for non-smokers were measured in offices, one indoor environmental category in which significant exposure might be expected to occur (3).

### EXPERIMENTAL

#### Selection of Offices

The primary criterion determining selection was that the office space to be sampled be shared by two or more persons of whom at least one smoked. None of the businesses involved in the survey had any type of mandated smoking policy. However, in some offices certain areas were recognized for smoking, either by agreement or habit. An independent organization selected the offices for the survey. Candidate offices were contacted by telephone. The selection process also was directed by guidelines that were designed to vary the sample of offices with respect to: nature of business, age of building, number of floors in building, number of employees in office, and size of office space sampled. Additional criteria related to the availability of adequate sampling locations. Contacts at offices were given the freedom to designate the office space to be sampled in cases where more than one location in the office

2023380251

meeting the selection criteria was available. Thirty-one offices were selected for the survey.

#### Sampling Methods and Procedure

Portable air sampling systems (PASS's) (4) were used for the measurements. The sampling procedures employed were designed to collect air samples for determining the concentrations of vapor phase nicotine, ultraviolet particulate matter (UV-PM), respirable suspended particles (RSP), and to monitor the concentration of carbon monoxide (CO) and the air temperature and barometric pressure. Sampling procedures utilized established personal exposure monitoring equipment and techniques that were designed to be unobtrusive. One set of measurements (nicotine, UV-PM, RSP, and CO) was collected as an area sample in each office. The duration of each sampling period was about 60 minutes, during which the PASS was not moved.

The PASS is an area sampling device, powered by batteries and designed to appear as an ordinary briefcase. This design reduces the possibility that sampling will influence occupants' ordinary activities. During operation, the PASS remains closed and makes little noise. The briefcase's exterior has an on-off switch positioned under the handle and inlet and exhaust ports positioned diametrically at the corners. These items are brass and therefore match the briefcase's original hardware.

The specific sampling location within each office was chosen to obtain a sample that was representative of the air in the mixing zone of the room and depended on the actual floor plan and furnishings. Guidelines for selecting sampling locations were derived from those described by Nagda and Rector (5). In general, PASS locations were: near the centers of occupied areas; at least two feet away from walls, floors, and ceilings; at positions not directly affected by direct ventilation sources such as doors or air vents; and at places allowing sampling to be as unobtrusive as possible. A typical location was on a table, desk, or filing cabinet near the center of the room. A previously identified contact person was the only one aware that air sampling was being performed. Sampling was conducted during regular office hours (8:00 a.m. to 5:00 p.m.).

The PASS operator documented smoking activities by recording at five-minute intervals the number of cigarettes being smoked. The total number of cigarettes smoked was determined from these observations. Other recorded observations included the room size, PASS location, location of ventilation sources, and perceptions regarding the extent of air mixing and ventilation. When time permitted, outdoor CO concentrations were measured either immediately before or after collection of corresponding indoor samples.

The method employed for sampling nicotine was based upon the method used by the U.S. National Institute for Occupational Safety and Health (NIOSH) (6) and has been described (7). Major components of the nicotine sampling system include a sorbent tube containing XAD-4 resin that is connected with rubber tubing to a constant-flow sampling pump operated at a nominal flow rate of  $1 \text{ L} \cdot \text{min}^{-1}$ . Sorbent tubes extended two centimeters from the side of each briefcase; air samples were introduced directly into the tubes. Samples of particulate matter were collected with a system comprising an impactor separating at 3.5 microns ( $\mu\text{m}$ ), a filter cassette containing a 37-millimeter diameter  $1.0 \mu\text{m}$  Fluoropore membrane filter, and a constant-flow sampling pump operated at a nominal flow rate of  $2 \text{ L} \cdot \text{min}^{-1}$ . The inlet to the filter cassette was located immediately downstream of the impactor. Pumps associated with the nicotine and particulate matter sampling systems were obtained from SKC Inc., Eighty-four, PA.

CO concentrations were measured by pulling air through an electrochemical detector at a nominal flow rate of  $0.5 \text{ L} \cdot \text{min}^{-1}$ . CO monitoring systems were developed by modifying commercially available, passive sensors to operate with sampling pumps. CO detectors (obtained from Neotronics, N.S., Gainesville, GA) were fitted with sampling lines and sampling pumps (obtained from Gilian, Inc., Wayne, NJ). The system also

2023380256

2023380257

included a voltage regulated power supply to the pump in order to maintain constant flow to the sensor. The detector was interfaced to a Model 21X data logger obtained from Campbell Scientific, Inc., Logan, UT. For use in the field, the data logger was programmed to record data once per minute. Sampling times also were recorded by the data logger, which is interfaced to the on-off switch. Recorded data were transferred to personal computer for interpretation.

Daily calibrations were performed on particulate matter and nicotine sampling pumps. Pump flow rates were measured with a primary standard (a bubble meter), then converted from actual to standard conditions of temperature and pressure (25°C and 760 torr). Pumps were adjusted when flow rates deviated more than 10% from respective nominal flow rates. Measured flow rates, as distinguished from nominal flow rates, were used for computing concentration results.

A calibration curve was prepared for each CO analyzer with certified standards of CO in air contained in cylinders acquired from Scott Specialty Gases, Plumsteadville, PA. Concentrations of the standards were 0.9 and 40 ppm CO. The calibration curves were used to adjust the CO measurements obtained during sampling.

#### Analysis:

Nicotine was analyzed according to the method described by Ogden *et al.*, (7). Nicotine collected on the XAD-4 resin was desorbed in 2 ml ethyl acetate containing 0.01% (v/v) triethylamine, which serves to neutralize acidic sites on analytical glassware surfaces. Analyses were performed with a Tracor Model 565 gas chromatograph equipped with a nitrogen-phosphorus detector. Chromatography was accomplished with a 30 m x 0.53 mm inside diameter, fused silica capillary column coated with a 1.5 micron film of DB-5 (5% phenyl methylpolysiloxane, obtained from J & W Scientific, Inc., Folsom, CA). Quinoline (Aldrich Chemical Company, Milwaukee, WI) was employed as an internal standard. For each sample, the rear segment of XAD-4 resin was analyzed separately in order to assess breakthrough, of which none was observed. Desorption efficiency was quantified according to the procedure contained in the NIOSH method (6). Six blanks were analyzed in conjunction with the field samples.

An attempt was made to quantify RSP according to the method described by Conner *et al.*, (8). Filters and samples were humidified at room temperature above a 50% aqueous solution of ethylene glycol for at least 12 hours before initial or final weighing. Static charges were removed by holding filters and samples under an antistatic device (Staticmaster, Model No. 2V500, Nuclear Products Co., El Monte, CA) for at least one minute prior to each weighing. Weights were measured with a Cahn 21 microgram balance. An antistatic device also was attached to one of the balance's interior walls. Each gravimetric result was the mean of five separate weighings.

UV-PM was quantified with the method described by Conner *et al.*, (8). This method utilizes the same samples as the method for determining RSP. After RSP is measured, the sample is extracted with 4 ml methanol and a 50  $\mu$ l aliquot is injected into a columnless liquid chromatographic system equipped with an ultraviolet detector measuring sample absorbance at 325 nm. Masses of UV-PM are then interpreted with a standard calibration curve derived from known concentrations of ETS generated in an environmental chamber (9). For the work reported here, methanolic solutions of 2,2',4,4' tetrahydroxybenzophenone (Aldrich Chemical Company, Milwaukee, WI) were employed as secondary standards. Six blanks were analyzed for RSP and UV-PM in conjunction with the field samples. Ingebrethsen *et al.*, (10) have reported results from comparative evaluations performed in the environmental chamber that show the RSP and UV-PM methods are unbiased relative to piezoelectric balances. Ogden *et al.*, (11) have reported results of collaborative tests of the methods for determining nicotine, RSP, and UV-PM.

## RESULTS

Results from the survey are presented in Table I. Concentrations of nicotine and UV-PM are at standard conditions of temperature and pressure (25°C and 760 torr). The mean sampling time was 65 min. The tabulated carbon monoxide concentration differences, delta CO, are computed relative to ambient concentrations by subtracting outdoor measurements from corresponding indoor measurements. Nicotine was not detected for samples 1, 14, and 28; the concentration limits shown for these samples are computed from respective volumetric data and the analytical limit of detection, 0.1  $\mu\text{g}$ . For eight sample locations, outdoor samples for carbon monoxide were not obtained; in the table these and the corresponding data entries are identified by NA, "not available".

Measurement results are summarized in Table II. The concentrations of nicotine, UV-PM, and CO represent the mean levels that non-smokers were exposed to in the 31 Ottawa offices. Statistically, the measurements are log-normally distributed and better represented by the geometric mean. For example 79% of the nicotine measurements were less than the arithmetic mean ( $12 \mu\text{g} \cdot \text{m}^{-3}$ ) while 54% were less than the geometric mean ( $7.2 \mu\text{g} \cdot \text{m}^{-3}$ ). This is also the case for the UV-PM measurements, with 81% less than the arithmetic mean ( $44 \mu\text{g} \cdot \text{m}^{-3}$ ) and 52% less than the geometric mean ( $24 \mu\text{g} \cdot \text{m}^{-3}$ ). The CO measurements also exhibit similar distributions. Note that those measurements below limits of detection were excluded from the statistical calculations for all cases (nicotine, UV-PM, and CO).

Six blank tubes and filters were analyzed for nicotine and UV-PM along with other samples. These blanks were carried into the field and handled in the same manner as the other samples with the exception that no air was pulled through them. No nicotine was measured in any of the blank XAD-4 tubes at the detection limit of the method (0.1  $\mu\text{g}$ ). The average background value in the blank particulate filters (1.5  $\mu\text{g}$ ) was used to adjust, or "blank correct", the UV-PM sample values.

RSP values generated from this survey are not reported. Absolute weights of particulate matter samples collected on the filters were near the detection limit of the method as estimated from the variation of the weights of the filter blanks. The result of this was that individual RSP sample differences could not be quantitatively determined. With the sampling and analytical techniques used, it would have been necessary to sample longer than 60 minutes to collect enough particulate matter to measure RSP quantitatively.

## DISCUSSION

Exposures represented by the measured concentrations of nicotine and UV-PM may be placed in perspective by presenting them in terms of cigarette equivalents. This type of interpretation has been used by other researchers for ETS (12,13,14). As used here, this measure is intended strictly as an estimate of exposure as distinguished from dose. Assumed for the calculations are a breathing rate of  $8.6 \text{l} \cdot \text{min}^{-1}$  and a cigarette delivering 0.96 mg nicotine and 12.2 mg "tar". The breathing rate corresponds to that associated with "miscellaneous office work" (15). The nicotine and "tar" values are sales weighted averages for cigarettes sold in Canada in 1986 (16). Also assumed for the calculations is the equivalence of UV-PM and "tar". Results from these calculations are presented in Table III. Based on the results of this survey, the average office worker was exposed to 0.0039 cigarette equivalent per hour (using nicotine as a marker) 0.0010 cigarette equivalent per hour (using UV-PM as a marker). Put another way, the time for exposure to one cigarette equivalent would have been 260 hours (using nicotine) or 1000 hours (using UV-PM).

The nicotine concentration results are comparable to results reported previously. (Sterling *et al.*, (17) have reviewed the literature relative to ETS measurements up to 1981). Muramatsu *et al.*, (18) used personal sampling techniques involving silicone OV-17 as the sorbent medium. These researchers collected samples in three

2023380258

TABLE I

**RESULTS OF NICOTINE, ULTRAVIOLET PARTICULATE  
MATTER, AND CARBON MONOXIDE MEASUREMENTS  
IN OTTAWA OFFICES (AUG. 25 - 28, 1987)**

SAMPLE NUMBER	SAMPLING TIME (MIN)	NO. OF CIGARETTES SMOKED	NICOTINE CONC. μg m⁻³	UV-PM CONC. μg m⁻³	CARBON MONOXIDE CONCENTRATION (ppm)		
					INDOOR	OUTDOOR	DELTA <sup>a</sup>
1	85	1	<1.2	9	<0.1	0.5	-0.5
2	78	6	7.8	20	1.7	0.4	1.3
3	62	3	5.6	25	0.6	1.6	-1.0
4	60	4	4.6	21	<0.1	<0.1	0.0
5	74	1	1.7	7	0.6	0.1	0.5
6	61	2	9.1	24	1.2	NA	NA
7	73	1	4.1	22	3.1	3.0	0.1
8	64	4	8.2	15	1.1	0.7	0.5
9	80	4	7.4	14	1.1	4.0	-2.9
10 <sup>b</sup>	60	5	69.7	104	3.4	0.4	3.0
11	56	1	2.4	26	0.4	0.5	-0.1
12	62	9	7.9	25	2.0	5.8	-3.8
13	84	2	1.7	14	0.7	<0.1	0.7
14	61	1	<1.6	12	1.4	NA	NA
15	61	4	4.1	6	1.8	NA	NA
16	65	2	3.7	15	0.9	5.3	-4.3
17	61	3	13.4	45	0.1	0.4	-0.3
18	60	2	11.1	30	<0.1	0.2	-0.2
19	53	2	2.0	7	0.2	1.4	-1.2
20 <sup>c</sup>	58	7	21.7	44	4.9	1.1	3.8
21	60	2	4.9	12	0.5	NA	NA
22	74	2	6.7	77	1.7	NA	NA
23	63	1	6.3	33	2.5	NA	NA
24	59	16	3.1	12	0.4	NA	NA
25	71	6	4.8	18	1.3	2.4	-1.1
26	59	3	30.5	31	2.6	3.4	-0.7
27	61	5	7.2	34	2.5	2.8	-0.3
28	57	1	<1.7	7	0.8	2.4	-1.6
29	61	3	8.2	51	1.6	NA	NA
30 <sup>d</sup>	67	31	58.3	426	8.7	2.0	6.6
31	60	7	19.2	186	6.6	1.6	5.0

(a) INDOOR-OUTDOOR

(b) SMALL OFFICE; OCCUPIED BY 2 SMOKERS

(c) NEAR SMOKERS "BREAK" AREA

(d) OFFICE OCCUPIED BY 3 SMOKERS; NUMBER OF CIGARETTES INCLUDES ADJACENT OFFICES

2023380259

**Table II. Descriptive Statistics for Nicotine, Ultraviolet Particulate Matter, and Carbon Monoxide Measurements**

	NICOTINE ( $\mu\text{g m}^{-3}$ )	UV-PM ( $\mu\text{g m}^{-3}$ )	CARBON MONOXIDE CONCENTRATION (ppm)		
			INDOOR	OUTDOOR	DELTA
Mean (arith.)	12.0	44	1.9	1.9	0.2
Mean (geo.)	7.2	24	1.3	1.2	NA
Min.	<1.2	6	<0.1	<0.1	-4.3
Max.	69.7	426	8.7	5.8	6.6
N	28	31	28	21	19

**Table III. Descriptive Statistics for ETS Exposure Estimated from Nicotine and UV-PM Measurements**

	EXPOSURE (CIGARETTE EQUIVALENTS PER HOUR)	
	NICOTINE	UV-PM
Mean (geo.)	0.0039	0.0010
Mean (arith.)	0.0064	0.0019
Minimum	0.0009	0.0002
Maximum	0.038	0.018
N	28	31

2023380260

offices in Japan and reported average concentrations ranging from 5.9 to 19.8  $\mu\text{g} \cdot \text{m}^{-3}$ . Oldaker *et al.*, (19) measured nicotine with PASS's in 47 offices in New York City and found that the concentrations were distributed log-normally ranging from 1.0 to 16.3  $\mu\text{g} \cdot \text{m}^{-3}$  with a geometric mean of 4.3  $\mu\text{g} \cdot \text{m}^{-3}$ . Hammond *et al.*, (20), employing filters impregnated with sodium bisulfite to collect personal nicotine samples, reported nicotine concentrations ranging from 3.1 to 28.2  $\mu\text{g} \cdot \text{m}^{-3}$  for four non-smoking office workers.

UV-PM results are consistent with results found in a 1986 survey conducted in New York City (19). Accordingly, for that survey concentrations in 47 offices ranged from 17 to 258  $\mu\text{g} \cdot \text{m}^{-3}$  with a geometric mean of 38  $\mu\text{g} \cdot \text{m}^{-3}$ .

Indoor and outdoor CO results likewise are comparable to results reviewed (17); this comparability is also shown for the delta CO results, which some researchers have used to provide indications of ETS. Computation of delta CO values assumes that indoor CO sources other than ETS are absent and that outdoor measurements represent the CO background. Subtracting outdoor CO measurements from indoor CO measurements thus should give the ETS contribution to the indoor CO concentration. For the present investigation, delta CO values vary considerably, ranging from -4.3 ppm (outdoor CO concentration higher than indoor CO concentration) to 6.6 ppm (indoor CO concentration higher than outdoor CO concentration).

For most samples, the outdoor CO concentration was higher than the indoor concentration. For example, the outdoor concentration was higher than the indoor concentration for 13 of the 23 sets of measurements, while the indoor was higher than the outdoor for 9 sets of measurements. Although higher delta CO measurements accompanied some of the samples with higher concentration of nicotine or UV-PM (sample numbers 10, 20, 30 and 31), delta CO does not appear to be a useful marker for ETS because of variation in CO levels caused by other combustion sources.

Results from the thirty-one sampling sites investigated are expected to provide upper estimates of non-smoker exposure to ETS. All of the offices in which sampling was performed during this survey met the primary criterion, which was to sample in offices where active smoking was observed as the measurements were being taken. An unbiased and therefore presumably lower, estimate would have been obtained had samples been collected in randomly selected offices, because some of these would have been occupied only by non-smokers.

#### REFERENCES

1. U.S. Department of Health and Human Services, The Health Consequences of Involuntary Smoking, Rockville, MD, 1986.
2. Committee on Passive Smoking, Board on Environmental Studies and Toxicology, National Research Council, Environmental Tobacco Smoke. Measuring Exposures and Assessing Health Effects, National Academy Press, Washington DC, 1986.
3. D.J. Moschandreas, Bull. N.Y. Acad. Med., 57, 845-859 (1981).
4. G.B. Oldaker III, B.C. McConnell, P.F. Perfetti, R.F. Walsh, F.W. Conrad, Jr., B.J. Ingebrethsen, L.W. Eudy, M.W. Ogden and M.W. Stancill, presented at 41st Tobacco Chemists' Research Conference, Greensboro, NC, Oct. 1987.
5. N.L. Nagda and H.E. Rector, Guidelines for Monitoring Indoor Air Quality, EPA-600/4-83-046 (1983).
6. National Institute for Occupational Safety and Health, Manual of Analytical Methods, Part II, Vol. 3, Method No. S293, Cincinnati, OH, 1977.
7. M.W. Ogden, L.W. Eudy, D.L. Heavner, F.W. Conrad, Jr. and C.R. Green, presented at 40th Tobacco Chemists' Research Conference, Knoxville, TN, Oct. 1986.
8. J.M. Conner, J.M. Murphy, G.B. Oldaker III, C.R. Green and A.L. Angel, presented at 40th Tobacco Chemists' Research Conference, Knoxville, TN, Oct. 1986.
9. D.L. Heavner, F.A. Thome, L.W. Eudy, B.J. Ingebrethsen and C.R. Green, presented at the 79th Meeting of the Air Pollution Control Association, Minneapolis, MN, June 1986.

2023380261

10. D.J. Ingebretsen, D.L. Heavner, A.L. Angel, J.M. Conner, T.J. Steichen and C.R. Green, J. Air Pollut. Control Assoc., 38, 413-417 (1988).
11. M.W. Ogden and J.M. Conner, presented at 101st Meeting of the Association of Official Analytical Chemists, San Francisco, CA, Sept. 1987.
12. M. Muramatsu, S. Umemura, T. Okada and H. Tomita, Environ. Res., 35, 218-227 (1984).
13. J.L. Repace and A.H. Lowery, Science (Washington DC), 208, 464-472 (1980).
14. W.C. Hinds and M.W. First, W. Engl. J. Med., 292, 844-845 (1975).
15. American Society of Heating, Refrigerating and Air Conditioning Engineers, Inc. ASHRAE Handbook, 1985 Fundamentals, Atlanta, GA, 1985, pp. 8.11-8.12.
16. G.B. Oldaker III, personal communication, 1988.
17. T.D. Sterling, H. Dimich and D. Kobayashi, J. Air Pollut. Control Assoc., 32, 250-259 (1982).
18. M. Muramatsu, S. Umemura, J. Fukui, T. Arai and S. Kira, Int. Arch. Occup. Environ. Health, 59, 545-550 (1987).
19. G.B. Oldaker III, J.M. Conner, F.W. Conrad, Jr., B.C. McConnell, M.W. Ogden, P.F. Perfetti, M.W. Stancill, R.A. Fenner and W.E. Crouse, presented at 41st Tobacco Chemists' Research Conference, Greensboro, NC, Oct. 1987.
20. S.K. Hammond, B.P. Leaderer, A.C. Roche and M. Schenker, Atmos. Environ., 21, 457-462 (1987).

#### ACKNOWLEDGMENTS

The authors gratefully acknowledge the assistance provided by Mr. Paul Doucet for his efforts in locating sampling sites. We also thank Guy B. Oldaker III, Terry L. Miller and Michael Barkdoll for their help and comments. This survey was funded by the Smokers' Freedom Society.

2023380262

# Environmental Tobacco Smoke and Indoor Air Quality in Modern Office Work Environments

Theodore D. Sterling, PhD; Chris W. Collett, MA; and Elia M. Sterling, BA, BArch

Recent attempts to clean the air in modern sealed office buildings appear to have focused on one component of indoor air quality, environmental tobacco smoke (ETS). Prohibiting smoking entirely or designating specific smoking areas has been suggested to improve comfort of office workers and reduce acute symptoms of so-called "building illness." The effectiveness of such methods, as well as the overall relation of ETS to indoor air quality, are here evaluated, based on reviews of a large number of studies of indoor air quality in modern office buildings under normal use and occupancy. Under these conditions, ETS does not appear to contribute significantly to a build-up of contaminants in offices. Also, in two large series of studies of buildings with health and comfort complaints in the US and Canada, ETS does not appear to be associated with cases of building illness.

Pollutants may build up inside the modern office space. They are generated by a wide variety of sources, both in and out of the building, and may be the cause of occupant discomfort and illness. Attention has been focused on one component of indoor air quality: environmental tobacco smoke (ETS). Reasons for that attention are not hard to find. Smoking has been associated with a large number of diseases, and tobacco smoke is the most visible indoor source of combustion by-products. On the other hand, there are many sources emitting toxic materials indoors, and modern sealed office buildings tend to generate, entrap, concentrate, and circulate a large number of aerosol contaminants

so that the indoor atmosphere may become highly polluted in the absence of smoking.<sup>1-10</sup> These circumstances give rise to two questions: (1) to what extent is the overall quality of the air in the office workplace affected by smoking workers, and (2) to what extent is ETS associated with health and comfort complaints of office workers in modern, sealed, so-called energy-conserving buildings?

Based on a large number of investigations, much is now known about the relation of ETS to indoor air quality and of outbreaks of building illness. It is our purpose to review the contribution of ETS to overall indoor air quality in the modern office environment under conditions of normal use and occupancy. Our review is based on the results of extensive field monitoring that has been undertaken since the mid-1970s by investigators from both government and private sectors. (All investigations referred to here that are not published in journals are available on request from agencies doing the studies or from principal investigators.) We will briefly treat three distinct but related issues: (1) sources and concentrations of pollutants in modern office buildings; (2) environmental tobacco smoke as a component of indoor air quality; and (3) building-related illness and indoor air quality.

## Sources and Concentrations of Pollutants in Modern Office Buildings

From the School of Computing Science, Faculty of Applied Sciences, Simon Fraser University, Burnaby, British Columbia V5A 1S6 Canada (address correspondence to Dr. Sterling, Professor); and Theodore D. Sterling Ltd., 1507 W. Twelfth Ave., Vancouver, British Columbia V6J 2Z2, Canada (Mr. Collett and Mr. Sterling).

0096-1736/87/2901-07\$08.00/0

Copyright © by American Occupational Medical Association

A wide range of substances has been measured inside modern office buildings. Table 1<sup>1</sup> lists the major groups of indoor contaminants and gives their sources, subdivided into three groups, based on the location of their sources. Group A lists those with predominantly outdoor sources; Group B, with sources both indoors and outdoors; and Group C, with predominantly indoor sources.

2023380263

It is evident that the majority of indoor pollutants come from multiple sources, both indoors and outdoors.

A wide range of contaminants has been measured as part of indoor air quality investigations of offices and other public buildings since the mid-1970s. The work has been done by a large number of investigators from the public and private sector, with the majority coming from the United States. In order to make more general use of these study results, relevant air quality and other data collected by these investigators have been extracted directly from their reports and stored in a Building Performance Database (BPD) developed by these authors.<sup>3,4</sup> A similar data base, "Concentration of Indoor Pollutants Data Base" (CIPDB), limited to buildings in the United States and concentrating more on residences than on offices, has been developed by the

Lawrence Berkeley Laboratory at the University of California.<sup>5</sup> The BPD currently contains information from 230 investigations of office buildings throughout North America and Western Europe, and includes reported concentrations for 189 different pollutants. The CIPDB currently contains summary data for 192 reports, including nine different pollutants.

Table 2 presents data contained in BPD of both indoor and outdoor concentrations of the 12 pollutants most frequently measured in indoor air quality investigations of office buildings. The number of measurements, the median concentrations, and the range of values are shown for each pollutant. Measurable concentrations of indoor and outdoor-generated pollutants were often found to be present in modern office buildings. Only carbon dioxide (CO<sub>2</sub>) and formaldehyde concentrations

TABLE 1  
Indoor Air Pollutants by Source\*

Group A. Source predominantly outdoor	
Cadmium	Industrial emissions, suspensions of soils
Calcium, chlorine, silica	Automobiles
Lead, manganese	Photochemical reactions
Ozone	Diesel fuel combustion, industrial emissions
Sulphur oxides	
Group B. Sources both indoor and outdoor	
Carbon dioxide	Metabolic activity, combustion
Carbon monoxide	Combustion
Nitrogen oxides	Combustion
Organic substances	Petrochemical solvents, vaporization of unburned fuels, paint; metabolic activity, pesticides, insecticides, fungicides, adhesives, household solvents, cooking, cosmetics
Particulates	Combustion, condensation, human skin and hair, carpet, shampoo
Polycyclic hydrocarbons	Automobile exhausts and tobacco smoke
Viable organisms, allergens, and pollen	Fungi, molds, bacteria, viruses, dust, animal dander, trees, grass, plants
Water vapor	Biological activity, combustion, evaporation
Group C. Sources predominantly indoor	
Aerosols	Consumer products
Ammonia	Metabolic activity, cleaning products
Asbestos, mineral and synthetic fibers	Fire-retardant, acoustic, thermal, or electrical insulation
Formaldehyde and other aldehydes	Particle board, insulation, furnishings, combustion

\* Adapted from the National Academy of Sciences/National Research Council 1981.<sup>1</sup>

TABLE 2  
Comparison of Indoor/Outdoor Pollutant Levels From 230 Studies Contained in Building Performance Database

Pollutant	Indoor Concentrations			Outdoor Concentrations		
	No. of Measurements	Median	Range	No. of Measurements	Median	Range
Aldehydes*	10	ND†	ND-0.03 mg/m <sup>3</sup>	—	—	—
Amines	9	ND	ND-404 ppb	—	—	—
Aromatic hydrocarbons	118	Trace	ND-104 mg/m <sup>3</sup>	8	0.0125 mg/m <sup>3</sup>	ND-2.5 mg/m <sup>3</sup>
Carbon dioxide	100	509.6	ND-2300 ppm	19	375 ppm	190-525 ppm
Carbon monoxide	209	3.1	ND-242 ppm	35	2 ppm	ND-32 ppm
Formaldehyde	207	0.013 ppm	ND-0.6 ppm	24	0.001 ppm	ND-0.031 ppm
Hydrocarbons	129	Trace	ND-28 ppm	12	0.032 ppm	ND-8 ppm
Nitrogen oxides	97	ND	ND-160 ppb	17	27.5 ppb	ND-570 ppb
Nitrogen dioxide	45	ND	ND-100 ppb	7	7 ppb	ND-67 ppb
Ozone	82	ND	ND-95 ppb	9	12 ppb	ND-100 ppb
Particulates	101	0.038 mg/m <sup>3</sup>	ND-0.7 mg/m <sup>3</sup>	47	0.037 mg/m <sup>3</sup>	ND-0.091 mg/m <sup>3</sup>
Sulfur dioxide	40	ND	ND-0.17 ppm	5	—	ND-0.17 ppm
Temperature	112	73°F	61-80.8°F	24	61°F	41-88°F
Relative humidity	114	39.1%	4-74%	21	57%	22-98%

\* Not including formaldehyde.

† ND, nondetectable level.

2023380264

were substantially higher indoors than outdoors. Carbon monoxide (CO) concentrations were slightly higher indoors, whereas ozone and nitrogen oxides were lower indoors than out. All other concentrations of commonly measured pollutants differed very little between indoors and outdoors. Similar findings were reported in a number of published reviews of indoor/outdoor pollutant relationships.<sup>1-7</sup>

#### Environmental Tobacco Smoke as a Component of Indoor Air Quality

Combustion byproducts produced by the burning of tobacco products include a wide range of chemical constituents: carbon monoxide, particulates, nitrogen oxides, aromatic hydrocarbons, acrolein, aldehydes (including formaldehyde), nicotine, nitrosamines, hydrogen cyanide, and ketones.<sup>1-8</sup> The combination of the chemical constituents is referred to as environmental tobacco smoke (ETS).

Many of the field studies of indoor air quality in public buildings provide data for comparisons of pollutant concentrations measured in smoking and nonsmoking office buildings under normal conditions of use and operation. Table 3 compares measurements taken in office areas where smoking was permitted with office areas where smoking was restricted. Table 3, similar to Table 2, provides the number of measurements and the medians and ranges of concentrations found.

Table 3 shows that several of the constituent by-products of ETS, including carbon monoxide, particulates, nitrogen oxides, aromatic hydrocarbons and aldehydes, have been widely monitored in public buildings. Under normal conditions of ventilation and occupancy, the concentration of pollutants appears to vary little between office areas where smoking is permitted and where it is not. For example, in 909 measurements of

office buildings, median levels of CO were 3.1 ppm in smoking-permitted areas and 3.4 ppm in smoking-restricted areas. For all practical purposes, these are identical concentrations. In 101 measurements in office buildings, median particulate concentrations actually were found to be the same (0.038 mg/m<sup>3</sup>) for office areas where smoking was permitted and where it was restricted.

The only contaminant for which median concentration varied significantly between office areas where smoking was permitted and where it was not was CO. Although CO<sub>2</sub> is also considered a component of ETS, its main source by far is human metabolic activity.<sup>9</sup> In 100 measurements in office buildings, the median level of CO<sub>2</sub> was found to be substantially higher in nonsmoking office areas (759.4 ppm) than in areas where smoking was permitted (506.5 ppm). With the exception of CO<sub>2</sub>, then, there does not appear to be a substantial difference in contaminant concentrations between offices where smoking is restricted and where it is not. These findings should not be confused with concentrations of tobacco by-products (ETS) measured in restaurants, bars, nightclubs, sport arenas, waiting rooms, crowded lobbies, various modes of transportation, and experimental research chambers where very high rates of smoking often combine with poor ventilation. When increased cigarette consumption is combined with poor or no ventilation, levels of ETS constituent increase.<sup>6-10</sup>

#### Nonsmoker Exposure to ETS in the Office

A number of studies provide information on the rates of smoking and general smoking practices under normal working conditions, as well as the concentrations of particulates (total or respirable) in the building monitored<sup>6-11,12,13</sup> (D. Sterling, personal communication, 1986; unpublished data, 1983 and 1985). Results from

TABLE 3  
Comparison of Pollutant Levels in Buildings Where Smoking Is Permitted and Where Smoking Is Restricted From 230 Studies Contained in Building Performance Database

Pollutant	Smoking Permitted			Smoking Restricted		
	No. of Measurements	Median	Range	No. of Measurements	Median	Range
Aldehydes*	10	ND†	ND-0.03 mg/m <sup>3</sup>	—	—	—
Amines	6	ND	ND-404 ppb	3	ND	ND
Aromatic hydrocarbons	112	Trace	ND-12.5 mg/m <sup>3</sup>	6	0.012 mg/m <sup>3</sup>	ND-104 mg/m <sup>3</sup>
Carbon dioxide	94	506.5 ppm	ND-2300 ppm	6	759.4 ppm	ND-2000 ppm
Carbon monoxide	194	3.1 ppm	ND-242 ppm	15	3.4 ppm	ND-75 ppm
Formaldehyde	200	0.016 ppm	ND-0.6 ppm	7	ND	ND-0.22 ppm
Hydrocarbons	124	Trace	ND-28 ppm	5	ND	ND-Trace
Nicotine	10	8.5 µg/m <sup>3</sup>	ND-53.01 µg/m <sup>3</sup>	—	—	—
Nitrogen oxides	92	ND	ND-160 ppb	5	26.5 ppb	ND-70 ppb
Nitrogen dioxide	43	2 ppb	ND-100 ppb	2	ND	ND
Ozone	76	ND	ND-90 ppb	6	18 ppb	ND-95 ppb
Particulates	81	0.038 mg/m <sup>3</sup>	ND-0.7 mg/m <sup>3</sup>	20	0.038 mg/m <sup>3</sup>	0.014-0.32 mg/m <sup>3</sup>
Sulfur dioxide	39	ND	ND-0.17 ppm	1	ND	ND
Temperature	108	73°F	61°-80.8°F	4	74.5°F	72.75°-80°F
Relative humidity	114	38.1%	4-74%	4	62.25%	16-72%

\* Not including formaldehyde.

† ND, nondetectable level.

2023380265

the various studies show an average of two to three cigarettes per smoker per hour per work site in office buildings in Great Britain; rates of two to six and two to three cigarettes per smoker per hour per work site for a number of office buildings in the midwestern United States. (A work site usually includes five to ten workers.)

In two of the studies, particulate concentrations were measured immediately before a cigarette had been lit and during the time of smoking<sup>11</sup> (unpublished data, 1988). The two studies provide similar data showing a mean increase in particulate concentrations measured at all work sites of 0.003 mg/m<sup>3</sup>/cigarette, with a range of 0.002 to 0.0035 mg/m<sup>3</sup>/cigarette. Both studies show particulate concentrations again declining at the cessation of smoking. Although smoking thus increases particulate concentrations, the slight increase does not appear to significantly influence the average indoor concentrations under normal ventilation practices.

In addition to the relative contribution of tobacco smoke to indoor air pollution, data are now available to determine the proportion of office occupants who are nonsmokers and the proportion of these nonsmokers who are actually exposed to ETS. Data from the National Health Interview Survey (NHIS) in the United States and the Canadian Health Survey (CHS) show that approximately 70% of people in white-collar occupations in North America are nonsmokers.<sup>14,15</sup> Other research has estimated that less than half of nonsmokers are actually exposed to ETS in their workplace.<sup>16,17</sup>

Overall, the available data from research in the modern office environment show that, despite an appreciable number of smokers in offices, the presence of ETS does not significantly increase the indoor concentration of pollutants under normal conditions of building ventilation.

#### Building-Related Illness and Indoor Air Quality

Within the last decade, health and comfort complaints among occupants of modern, sealed office buildings related to indoor climatic conditions have been reported for hundreds of buildings throughout North America and Europe.<sup>18</sup> The types of complaints are surprisingly similar and range from headache and eye irritation to reproductive system and pregnancy problems. These symptoms are often associated with uncomfortable environmental conditions such as the air being too cold or too dry, work sites being too drafty or too stuffy, and the presence of odors in the work site. Buildings with such problems are now popularly referred to as sick buildings, and the epidemic of complaints from occupants of these buildings has been defined by the World Health Organization as sick building syndrome,<sup>19</sup> but is better known as *building illness*. The broad spectrum of indoor air quality or, rather, indoor air pollution has been considered by many researchers to be the principal cause of symptoms and thus has been monitored carefully where problems occur.

Since 1978, the US National Institute for Occupational

Safety and Health (NIOSH) has conducted more than 350 investigations of buildings with health and comfort problems. The findings from 903 of these investigations undertaken through 1983 were reviewed and tabulated by the Health Hazard Evaluation Branch of NIOSH.<sup>20</sup> Table 4 lists the suspected causes of the indoor air quality problems documented through 1983. Cigarette smoking was suggested as a suspected cause in only 2% of the investigations. By far the most prevalent problem was that of inadequate ventilation, with nearly half (48.3%) of the investigations attributing indoor air quality problems to this factor. Ventilation is deemed inadequate when the amount of fresh (outside) air is insufficient to dilute the level of indoor contaminants. Ventilation standards, in fact, specify minimum levels of fresh air reaching occupants. The most common cause of inadequate ventilation is the diminished intake of fresh air into the air circulation system, usually to conserve energy and save on cost of building operation. (In cold areas, intake air has to be heated and, in hot

TABLE 4  
Suspected Causes of Problems From Health Hazard Evaluation Branch  
National Institute for Occupational Safety and Health<sup>a</sup>

No.	% of Total
Inadequate ventilation	98 48.3
Contamination (inside)	36 17.7
Contamination (outside)	21 10.3
Humidity	9 4.4
Contamination (building fabrc)	7 3.4
Hypersensitivity pneumonitis	6 3.0
Cigarette smoking	4 2.0
Noise/Illumination	2 1.0
Scabies	1 0.5
Unknown	19 9.4
Total	203 100.0

<sup>a</sup> Adapted from Melius et al. 1984.<sup>20</sup>

TABLE 5  
Suspected Causes of Problems From Health and Welfare Canada Medical Services Branch Indoor Air Quality Investigations<sup>b</sup>

No.	% of Total
Inadequate ventilation	64 68
Poor air circulation	
Inadequate outdoor air (CO <sub>2</sub> > 800 ppm)	
Poor temperature/humidity control	
Outdoor contaminant	9 10
Reentry of building exhaust	
Motor vehicle exhaust	
Indoor contaminant	5 5
Copy machines	
Tobacco smoke	
Building fabric	2 2
Glues and adhesives	
Formaldehyde and organics	
Biological contaminants	0 0
No problem identified	14 15
Total	94 100

<sup>b</sup> Adapted from Kirkbride 1985.<sup>21</sup>

2023380266

areas, intake has to be cooled.) Other causes are malfunctions of airflow mechanisms or stratification of air, a condition in which a large part of the fresh circulated air travels along the ceiling and fails to mix fully with air at the breathing zone.<sup>21</sup> Varied contaminant sources from inside or outside were the cause of problems in 30% of the investigations. Many of them related to poor designs of ventilation systems (such as air intake and infiltration from garages or congested traffic areas). In 10% of the investigations, the causes of problems were determined as noise, illumination, or humidity. For the final 10% of the investigations, causes could not be determined. A similar distribution of causes of indoor air quality problems was determined by a more recent investigation by NIOSH of another 150 buildings (J. Carpenter, personal communication, 1986). These findings agree with a review of apparent causes of building illness by the staff of Health and Welfare Canada (HWC).<sup>22-23</sup> HWC is engaged in a health hazard evaluation program that now includes indoor air quality. A recent review of 94 building investigations (see Table 5) finds problems with the ventilation system in 64 instances, reentry of building or entry of motor vehicle exhaust in nine cases, combined problems with photocopy machines and tobacco smoke in five cases, and emissions from glues and adhesives in two instances.

Overall, results from the investigation of health and comfort complaints in sick buildings indicate a wide range of contributory causes of indoor air quality problems. Findings have not shown a consistent association between ETS and so-called sick buildings. By far, the most pervasive and consistent cause appears to be inadequate ventilation.

## Conclusions

A commitment by governments and professionals involved in the building process, building owners and operators, and office worker unions to improve the air quality in modern sealed office buildings is undoubtedly a worthwhile and well overdue objective. The provision of clean air is technically achievable. Prohibiting smoking entirely or designating specific smoking areas is often proposed as an effective means to provide clean air to office occupants.

Although any smoking restriction could very well affect the prevalence of smoking among office workers, prohibition of smoking has not been shown to have any measurable effect on either indoor air quality or associated health and comfort symptoms of sick building syndrome. Ventilation required to remove indoor contaminants produced by the occupants themselves, specifically CO<sub>2</sub> and body odor, will also remove the constituents of ETS. On the other hand, if adequate ventilation rates are not provided, then indoor-generated substances and dusts and chemicals infiltrating the building envelope from outdoors increase in concentration to unacceptable levels, even if ETS should be entirely absent.

Designation of special smoking areas might remove

multiple sources of irritation to smokers and nonsmokers alike. On the other hand, the segregation of smokers to specially designated smoking areas may have little effect and may well have undesirable impacts on ventilation performance. Concentrating smokers in designated smoking areas may place an excessive local burden on existing ventilation systems—a burden with which they may not have been designed to cope. The result may well be that smoking office workers are exposed to high levels of irritants for short time periods without any benefits accruing to their nonsmoking co-workers. To be of any real use, specially designated smoking areas may require installation and operation of higher volume ventilation systems with more effective air cleaning devices and possible direct venting outside.

## Acknowledgments

The Centers for Disease Control, Firemen's Fund Insurance, Lawrence Berkeley Laboratory, New York City Health Department, and the National Institute for Occupational Safety and Health made available the bulk of study reports from which data on indoor air contaminant concentrations were extracted for the Building Performance Data Base. Also, a number of colleagues contributed data from individual studies. Part of the grant support for our work comes as a special project from the Council for Tobacco Research, USA.

## References

1. Indoor Pollutants: National Academy of Sciences/National Research Council. Washington, DC: National Academy Press; 1981.
2. Sterling TD, Kobayashi D: Exposure to pollutants in enclosed "living spaces." *Environ Res* 1977;13:1-35.
3. Collett CW, Weinham JJ, Steeves JF, et al: The Building Performance Database: An analytical tool for indoor air quality research. Proceedings, Indoor Air Quality '86: Managing Indoor Air for Health and Energy Conservation. ASHRAE Conference, Atlanta, GA, in press 1986.
4. Sterling EM, Sterling TD: Pollutant concentrations in buildings. Proceedings, AIA Research and Design Conference, Los Angeles, CA, March 14-18, 1985.
5. Concentration of Indoor Pollutants Data Base (CIPDB). Users Manual. Building Ventilation and Indoor Air Quality Program, Berkeley, CA: Lawrence Berkeley Laboratory, 1985.
6. Sterling TD, Dimich H, Kobayashi D: Indoor byproduct levels of tobacco smoke: A critical review of the literature. *J Air Pollut Control Assoc* 1982;32:250-259.
7. Yocom JE: Indoor-outdoor air quality relationships. *J Air Pollut Control Assoc* 1982;32:500-520.
8. Turiel I: Indoor Air Quality and Health. Stanford, CA: Stanford University Press, 1985.
9. Hicks J: Tight building syndrome. *Env Health Occup* January 1984.
10. Repace JC, Lowrey AH: Indoor air pollution: tobacco smoke and public health. *Science* 1980;208:464-72.
11. Pelizzari ED: Indoor Air Quality Monitoring Programs Monthly Technical Progress Report No 2. EPA Contract 68-02-3679. Research Triangle Park, NC, 1983.
12. Hedge A: Building illness indices based on questionnaire responses. Proceedings, Indoor Air Quality '86: Managing Indoor Air for Health and Energy Conservation. ASHRAE Conference, Atlanta, GA, in press 1986.
13. Sterling EM, Sterling TD: Baseline data: Health and comfort in modern office buildings. Proceedings, 5th AIC Conference: The Implementation and Effectiveness of Air Infiltration Standards in Buildings, Reno, NV, Oct 1-4, 1984.

2023380267

14. National Health Interview Survey. (1979/1980 Data Tapes). Dept. of Health and Human Services. National Center for Health Statistics. Division of Vital Statistics. 1980.
15. Canada Health Survey. Health and Welfare Canada. Ottawa. 1978
16. Friedman GD, Poult DB, Balvoi RD. Prevalence and correlates of passive smoking. *Am J Public Health* 1983;73:401-5.
17. Arundel A, Irwin TE, Sterling TD, et al. Nonsmoker lung cancer risks from exposure to environmental tobacco smoke. In press. 1987
18. Berglund B, Lindvall T, Sandell J (eds). *Indoor Air*. Proceedings of the Third International Conference on Indoor Air Quality and Climate. Stockholm. Aug 20-23. 1984. vols 1-5.
19. Indoor Air Pollutants. Exposure and Health Effects Assessment. Working Group Report. Nordinen. Euro reports and studies no. 78. World Health Organization. Copenhagen. 1982.
20. Melius J, Wallingford K, Keenleyside R, et al. Indoor air quality. The NIOSH experience. Meeting of the American Congress of Government Hygienists. 1984
21. Hughes RJ, O'Brien D. Evaluation of building ventilation systems. *Am Ind Hyg Assoc J* 1986;47:807-213.
22. Watkins D. Indoor air quality in cold climates. hazards and abatement measures. *J Air Pollut Control Assoc* 1986;36:235-241
23. Kirkbride J. *Sick Building Syndrome. Causes and Effects*. Health and Welfare Canada. Ottawa. 1985.

P  
J  
R

M  
JC

### Penalty for Overconfidence

The wreck of the Titanic has at last been found, 580 miles off the coast of Newfoundland and in 13,000 feet of water. . . .

The vast luxury liner carried a load of millionaires and too few lifeboats. Its captain steamed it at full speed through an iceberg field of which he had been amply warned. Of 2,235 passengers and crew members, only 713 were saved.

...Gashed by an iceberg below the waterline, the ship . . . sustained a mortal wound. The water flowed over the top of the transverse bulkheads supposed to divide her into watertight compartments. Because the blow was so slight, and the officers gave no alarm, few passengers realized their danger. The ship's bow dipped gently into the water as the first lifeboats left, some half-empty. In one of them was my grandfather, a London high school teacher seeking fortune across the ocean.

Rowing away from the ship, he wrote in his account of sinking, he could see the lines of porthole lights ablaze, but bizarrely meeting the water at an angle. Gradually the angle increased until the great ship, one-sixth of a mile long, stood like a column in the moonlit sea.

There was a rumble as machinery crashed through the bulkheads, and the lights flashed out. Those in lifeboats watched the huge black shape slide silently into the icy waters. Then came the screams of the drowning, piercing the air for 40 minutes until, one by one, they died away.

The loss of the Titanic has served as a lasting reminder to the consequences of overconfidence. . . .

—From "The Editorial Notebook: The Titanic Lesson"  
by Nicholas Wade in *The New York Times*.  
Sept 4, 1985.

2023380268

# Concentrations of Nicotine, RSP, CO and CO<sub>2</sub> in Nonsmoking Areas of Offices Ventilated by Air Recirculated from Smoking Designated Areas

T.D. STERLING<sup>A</sup> and B. MUELLER<sup>B</sup><sup>A</sup>Faculty of Applied Sciences, Simon Fraser University, Burnaby, B.C. V5A 1S6 Canada;<sup>B</sup>Theodor D. Sterling Limited, #70 - 1507 West 12th Avenue, Vancouver, B.C. V6J 2E2 Canada

The exposure of nonsmokers to environmental tobacco smoke (ETS) when smoking is relegated to designated areas that are not separately ventilated is of considerable interest. Concentrations of nicotine, respirable suspended particles (RSP), carbon monoxide (CO), and carbon dioxide (CO<sub>2</sub>) were measured in offices under different conditions of smoking regulation: smoking prohibited; smoking prohibited areas receiving recirculated air from designated smoking areas; smoking and nonsmoking sections of these designated smoking areas. Nicotine was collected by pumping air for periods of 1-8 hr at 1 L/min through sampling tubes containing a styrene divinylbenzene copolymer. RSPs (5 µm cut-off) were measured using an optical side scattering instrument. CO was measured by a direct reading electrochemical analyzer and CO<sub>2</sub> by colorimetric detector tubes. Detection of nicotine in nonsmoking office areas that received recirculated air from smoking designated areas required sampling times of 4 hr or more. Nicotine levels in such offices were approximately 1.0 µg/m<sup>3</sup>. RSP, CO and CO<sub>2</sub> concentrations were approximately the same in these offices as compared to nonsmoking offices not exposed to recirculated air from smoking areas. Providing a designated but not separately ventilated smoking area appears to be effective in eliminating most components of ETS from nonsmoking office work areas.

## Introduction

A number of municipalities (San Francisco and Vancouver being leading examples) have passed bylaws to regulate smoking in public buildings. In principle these bylaws apply to public buildings and places of employment and establish a norm of no smoking except in smoking areas designated by the employer or proprietor. The Canadian and American Federal Governments are preparing to develop approaches to regulate smoking in workplaces under federal jurisdiction. Provincial and state governments are making similar preparations.

Four options are available to regulate office smoking:

1. Prohibiting smoking outright;
2. restricting smoking to designated areas that are ventilated separately;
3. restricting smoking to designated areas that are not ventilated separately; and
4. providing some framework by which an adjustment between smoking and nonsmoking workers may be achieved without directly regulating the placement of smokers.

The third option, that of providing a designated but not separately ventilated smoking area, appears to be the most frequently adopted procedure. A certain proportion of a building's population will demand a location where they may be allowed to smoke (for example, employees on their coffee and lunch breaks, members of the public waiting for services, or persons who are residents of the building—such as in prisons or hospitals.) Governments and the private sector own, operate and rent a wide variety of different

buildings, however, most of these buildings do not offer separate ventilation for different locations. To provide separate ventilation would not only be costly in many instances but very often physically impossible. Thus, the least disruptive and costly solution for many buildings appears to be the setting aside of designated but not separately ventilated smoking areas.

A question of considerable interest is the extent to which designated but not separately ventilated smoking areas are effective in decreasing exposure to environmental tobacco smoke (ETS) in nonsmoking areas. This project was designed to provide some information on that question.

The authors report here the outcome of a series of measurements of nicotine, respirable suspended particles (RSP), carbon monoxide (CO) and carbon dioxide (CO<sub>2</sub>) obtained in the following locations:

- 1) two cafeterias, each having smoking and nonsmoking areas;
- 2) four nonsmoking floors which received air recirculated from a ventilation system common to one of the cafeterias; and
- 3) two nonsmoking offices with independent ventilation systems which, therefore, did not receive air recirculated from designated smoking areas.

## Methods

Air sampling for nicotine, RSP, CO and CO<sub>2</sub> and an observation of the number of office occupants present and cigarettes smoked was undertaken in two adjacent buildings

2023380269

(Vancouver City Hall and City Hall Annex): Building A, which is a sealed, mechanically ventilated building, and Building B, which has opening windows and mechanical ventilation only in selected areas.

### Building Description

Building A is a 4-story sealed office building with 2 levels of underground parking. Each of the 4 floors contains approximately 1390 m<sup>2</sup> (15 000 ft<sup>2</sup>) of office space. Fresh air from an intake at ground level is supplied to an air-handling unit in the basement mechanical room. This fresh air is filtered, conditioned and then supplied unmixed to air induction units located at exterior walls. Air is returned, via ceiling return grates, to a second air-handling unit in the basement, which exhausts a portion of the return air, adds makeup air (minimum of 20%), and filters, conditions and returns the air to the occupied space via ceiling diffusers. As a result, indoor air from different parts of the building and different floors is mixed. Smoking is prohibited in all work areas and public areas of the building and is permitted only in the smoking section of the fourth floor cafeteria which is not separately ventilated.

Building B is a 12-story, unsealed building with opening windows and, originally, no mechanical ventilation system. Most areas are passively ventilated by building leakage while separate ventilation systems have been incorporated in only a few areas. In the offices where measurements were taken, rooms with exterior walls have opening windows. Additional ventilation is supplied to the central zone of each of these offices by an air-handling unit which receives fresh air from an intake at ground level. The zone air-handling unit feeds conditioned air to a supply-air plenum (in the ceiling space) where individual fan-coil units temper the air again and deliver it to the occupied space below. Air which has not been exhausted through windows or doors is returned to the ceiling plenum and again tempered by the fan-coil units. These systems, therefore, have no ducting common to other areas of the building. Smoking is prohibited in all work areas and public areas in the building except the smoking section of the cafeteria (which is located in the basement). Heated/cooled air is supplied separately to the cafeteria and exhausted through windows.

### Sampling and Occupant Observation

Three samples were taken in each of the smoking and non-smoking sections of the cafeterias of Buildings A and B; two samples on each of the four floors in nonsmoking offices of Building A; two samples in the nonsmoking offices of Building B; and two samples of RSP outdoors.

Samples for nicotine were obtained using a portable air sampling pump housed inside a briefcase. Because of the effect of air sampling on occupant behavior,<sup>11</sup> the sampling apparatus was designed to collect samples in an unobtrusive manner. Nicotine samples were collected by pumping air at 1 L/min through sorbent tubes containing XAD-4 resin, a styrene divinylbenzene copolymer. The sorbent tubes contained 80 mg of resin in the front (primary) section and 40 mg

in the rear (secondary) section. Samples were collected for 1 hr each in the cafeteria locations and for periods of 2, 4 or 8 hr at other sampling sites. Respirable suspended particles (5 µm cutoff) were determined using a P-5H digital dust indicator (Sibata Scientific Technology, Tokyo, Japan) which measures light side-scattered by suspended particles. The unit was calibrated at the factory to monodispersed stearic acid particles with a mean diameter of 0.3 µm. The unit usually measured respirable particles for the entire sampling period, depending on battery charge. Approximately midway into the 1- or 2-hr air sampling period, CO and CO<sub>2</sub> concentrations were measured at the sampling locations. (CO and CO<sub>2</sub> were measured more often during 4- and 8-hr sampling periods.) CO was measured using a direct-reading electrochemical analyzer (Nova 310 L, Nova Analytical Systems, Inc., Hamilton, Ontario) housed in a flight case. CO<sub>2</sub> was measured using colorimetric detector tubes (Gastec, Gastec Corporation, Yokohama, Japan) and a manual sampling pump.

During a sampling period, the number of occupants in a predefined observation area and the number of cigarettes smoked in that area were observed and recorded. The observation areas were defined by visual configurations and reliability of surveillance of the observable office area. For purposes of comparison, number of persons and cigarettes smoked were calculated per 10 m<sup>2</sup> where applicable. At the completion of sampling, the sorbent tubes were refrigerated until analysis.

### Analysis

In the chemical analysis of nicotine, resin beads in the sorbent tubes were transferred to gas chromatograph autosampler vials to which were added 50 µL of quinoline (100 mg/L) to serve as an internal standard and 1 mL of ethyl acetate as an extraction solvent. Triethylamine (0.01% by volume) was added to the extraction solvent to prevent adsorptive losses of nicotine onto the glass autosampler vials. Samples and spiked standards then were placed on an automatic shaking device and shaken for 30 min. A Hewlett-Packard Model 5880A or Model 5830A gas chromatograph equipped with a nitrogen-phosphorus detector was employed in conjunction with an autosampler and a GC terminal to determine peak areas of the nicotine and compare them with the areas obtained from nicotine standards. The assayed nicotine was corrected for the desorption efficiency (usually 94%) of the particular lot of XAD-4 resin used in sampling. Final nicotine results were divided by the volume of air sampled to yield results in µg/m<sup>3</sup>. The rear (backup) sections of sorbent tubes were analyzed separately and, except for one case, always yielded nicotine determinations less than the limit of detection, thus indicating no breakthrough of nicotine past the primary section. [The authors' procedure, by and large, is based on the National Institute of Occupational Safety and Health (NIOSH) method.<sup>12</sup>]

Respirable suspended particles were estimated by converting the digital counts of particles per sampling time to an average count per minute. A background count of 5 counts per min was subtracted from the average to yield RSP values in µg/m<sup>3</sup>.

2023380220

## Results

Table I summarizes measurements for RSP, CO, CO<sub>2</sub>, nicotine; average number of persons per 10 m<sup>2</sup>; and average number of cigarettes smoked per hour per 10 m<sup>2</sup> (where applicable). Because of the large variability and suspected skew of measures, means, medians and ranges are given. Measurements in the cafeteria smoking areas each are based on 6 samples as are measurements in the cafeteria nonsmoking areas. Because there were no perceptible differences between cafeterias in Buildings A and B, both for smoking and nonsmoking areas, their data have been merged. Measurements in nonsmoking office areas in Building A are based on 8 samples, and measurements in nonsmoking areas in Building B are based on 2 samples.

There were significantly more persons per unit area in the cafeterias than in the nonsmoking offices. The numbers of individuals per 10 m<sup>2</sup> in smoking and nonsmoking areas of cafeterias, however, were approximately the same. As might be expected, both CO and CO<sub>2</sub> levels were higher in the smoking than nonsmoking areas of the cafeterias. This also was true for RSPs. Nicotine levels averaged 14.0 µg/m<sup>3</sup> in the smoking area and 6.2 µg/m<sup>3</sup> in the nonsmoking area of the cafeterias. The drop in RSPs and nicotine from smoking to nonsmoking areas of the cafeterias is quite steep and attests to the rapid dilution of ETS.

Contributions to RSP, CO and CO<sub>2</sub> that are caused by smoking in the designated smoking area are diluted further in the recirculated air. This dilution can be seen from a comparison of measurements in the office areas of Building A with Building B. Concentrations of RSP, CO and CO<sub>2</sub> in Building A's nonsmoking areas, which received recirculated air from the smoking area, are approximately the same as those measurements taken in Building B, which did not receive any such recirculated air (also see Table II). Of special interest are measurements of nicotine. It is important to keep in mind that the detection of nicotine in air, in the

dilute quantities in which it may be present, requires a lengthy sampling procedure. As the concentration of nicotine in air decreases, larger air samples must be obtained to detect that concentration. For the method used here, a 2-hr sample at 1 L/min would detect nicotine concentrations greater than 0.8 µg/m<sup>3</sup>. Of 4 samples taken for 2 hr each, not a single sample detected a concentration above 0.8 µg/m<sup>3</sup>. For a 4-hr sample at 1 L/min, the lower level of detection is 0.4 µg/m<sup>3</sup>. At that level, 1 positive detection at a concentration of 1.0 µg/m<sup>3</sup> was made in 1 out of 3 samples. For the 1 sample taken for 8 hr, the lower level of detection was 0.2 µg/m<sup>3</sup>. That sample measured a concentration of 0.8 µg/m<sup>3</sup> (findings summarized in Table II).

## Discussion

Studies of office air quality have demonstrated that significant reductions in ETS related RSP may be achieved in nonsmoking areas when smoking is limited to designated areas that are not ventilated separately.<sup>(1)</sup> The extent of involuntary exposure to ETS, however, best may be established quantitatively when nicotine is used as the marker.\* It has been suggested<sup>(3)</sup> that advances in measurement technology may provide grounds for reliance on nicotine as a general indicator of ETS. Other components of ETS may be less useful for developing an ETS exposure index. ETS components are complex and variable and also include many constituents similar to those emitted from other sources.<sup>(4)</sup>

\*The observation that nicotine in sidestream smoke is mainly in the vapor phase while in mainstream smoke it is more in the particulate (deposit) phase poses no obstacle to the use of nicotine as an index of ETS infiltration because building occupants are not exposed to mainstream smoke unless they actively do smoke. The nicotine concentration obtained from sampling the air is a representative sample of ambient ETS inhaled by nonsmokers.

**TABLE I**  
**Comparison of ETS Related Air Quality Parameters in Nonsmoking Work Areas and Designated Smoking Areas**

		RSP <sup>A</sup> (µg/m <sup>3</sup> )	CO (ppm)	CO <sub>2</sub> (ppm)	Nicotine (µg/m <sup>3</sup> )	Persons /10 m <sup>2</sup>	Cigarettes /hr/10 m <sup>2</sup>
Smoking areas of Cafeterias A & B combined	Mean	70	3.9	690	14	1.8	1.2
	Range	23-129	1.1-11.4	450-1000	<1.6-43.7	0.79-3.42	0.53-1.67
	Median	74	2.5	650	11	1.6	1.2
Nonsmoking areas of Cafeterias A & B combined	Mean	32	2.6	560	6.2	1.7	
	Range	15-57	1.2-4.5	400-700	<1.6-10.9	0.76-2.5	NA <sup>B</sup>
	Median	26	2.4	580	7.9	1.7	
Nonsmoking office area, Building A	Mean	6	1.8	490	c	0.73	
	Range	4-11	1.3-2.3	400-580	c	0.28-1.9	NA
	Median	6	1.7	500		0.46	
Nonsmoking office area, Building B	Mean	7	1.35	450	c	0.9	
	Range	6-8	1.3-1.4	400-500	c	0.53-1.28	NA
	Median	7	1.35	450		0.9	

<sup>A</sup>Mean outdoor RSPs were 10 µg/m<sup>3</sup>.

<sup>B</sup>NA = not applicable

<sup>C</sup>See Table II

2023380271

**TABLE III**  
**Nicotine, RSP, CO and CO<sub>2</sub> Concentrations in Eight Locations in a No Smoking Office Area that Receives Recirculated Air from a Smoking Designated Area and in Two Locations without Such Recirculation<sup>A</sup>**

	Location	Sample Time (hr)	Nicotine ( $\mu\text{g}/\text{m}^3$ )	RSP ( $\mu\text{g}/\text{m}^3$ )	CO (ppm)	CO <sub>2</sub> (ppm)	Persons /10 m <sup>2</sup>
Recirculated air	1	2	<0.8	6	1.7	580	0.50
	2	2	<0.8	5	2.3	500	0.42
	3	2	<0.8	5	1.3	400	1.90
	4	2	<0.8	4	2.0	500	0.38
	5	4	<0.4	11	2.2	550	0.39
	6	4	<0.4	5	1.7	450	0.28
	7	4	1.0	6	1.7	500	1.02
	8	8	0.8	6	1.6	450	0.96
No recirculated air	9	B	B	8	1.4	400	0.53
	10	B	B	6	1.3	500	1.28

<sup>A</sup>Sampling time for nicotine ranged from 2 to 8 hr.

<sup>B</sup>Nicotine was not measured because these offices could not receive ETS from any source.

Air sampled for 2 hr at 1 L/min (using the NIOSH protocol) reliably measures nicotine levels that are larger than 0.8  $\mu\text{g}/\text{m}^3$ . Levels of nicotine appear to be at or below that concentration in offices in which smoking is prohibited but which receive air recirculated from smoking designated areas. To give meaning to such trace values, the exposure of an office worker to nicotine at 1  $\mu\text{g}/\text{m}^3$  for 1 hr can be calculated roughly. Given a breathing rate of 0.48  $\text{m}^3/\text{hr}$  for the level of activity required during normal office work,<sup>(5,6)</sup> an office worker would breath air containing 0.48  $\mu\text{g}$  of nicotine in 1 hr. This quantity is approximately equivalent to 1/1800 of the nicotine inhaled by actively smoking 1 cigarette (900  $\mu\text{g}/\text{cigarette}$ ).<sup>(6)</sup> Until relatively recently, calculations of a smoker's exposure to cigarette smoke was limited to amounts of materials in the mainstream smoke. Insofar as smokers are spatially close to their cigarette and often inhale relatively undiluted sidestream smoke, existing estimates of smokers' exposure to any component of ETS must be lower than their actual magnitudes. Thus, the nonsmoker probably inhales less than 1/1800 of the nicotine inhaled by a smoker when actively smoking one cigarette, unless this nonsmoker should be standing in very close proximity to a burning cigarette.

Based on these findings, it is the authors' belief that the provision of a designated smoking area appears to be effective in eliminating most traces of ETS from the rest of the office space, even if the designated smoking area is not separately ventilated. An exclusive reliance on regulating smoking while ignoring all other problems besides smoking which may influence the quality of air in the nonindustrial work environment may accomplish little in addressing indoor air quality problems, however, especially in so-called "sick buildings."<sup>(7)</sup>

If a designated area is made available for smoking in offices where otherwise smoking is not permitted, the designated space should be sufficiently large to prevent overcrowding.

#### Acknowledgment

Thanks are due to Mr. C. Collett for his help in arranging for sampling sites and to Mr. L. Strandebø for allowing access to the City Hall sampling sites. The authors are beholden to Dr. C. Nystrom for help with some of the equipment used for this work. Part of the costs of this project came from a special grant from the Council for Tobacco Research, Inc.

#### References

1. Lee, H.K., T.A. McKenna, L.N. Renton and J. Kirkbride: Investigation of the Impact of a New Smoking Policy on Office Air Quality in the National Library of Canada in Hull, Quebec. In *Proceedings of the 78th Annual Meeting of the Air Pollution Control Association*, Ottawa, 1985.
2. National Institute for Occupational Safety and Health: *Manual of Analytical Methods*. 2nd Ed. Cincinnati, Ohio: United States Department of Health, Education and Welfare/National Institute of Occupational Safety and Health, 1977. pp. S293-1-S293-9.
3. National Health and Welfare, Canada: Involuntary Exposure to Tobacco Smoke. Report of the Advisory Committee. Ottawa, Canada: Environmental Health Directorate, 1987.
4. Bayer, C.W. and M.S. Black: Passive Smoking: Survey Analysis of Office Smoking Areas vs. Environmental Chamber Studies. In *IAQ '86: Managing Indoor Air for Health and Energy Conservation*. Atlanta, Ga.: American Society of Heating, Refrigerating and Air-Conditioning Engineers, 1986. pp. 281-291.
5. American Society of Heating, Refrigerating and Air-Conditioning Engineers, Inc.: *ASHRAE Handbook: 1985 Fundamentals*. Atlanta, Ga.: American Society of Heating, Refrigerating and Air-Conditioning Engineers, Inc., 1985. pp. 8.11-8.12.
6. Miromatsu, M., S. Umemura, T. Okada and H. Tomita: Estimation of Personal Exposure to Tobacco Smoke with a Newly Developed Nicotine Personal Monitor. *Environ. Res.* 35:218-227 (1984).
7. Sterling, T.D., C.W. Collett and E.M. Sterling: Environmental Tobacco Smoke and Indoor Air Quality in Modern Office Work Environments. *J. Occup. Med.* 29:57-62 (1987).

5 November 1987; Revised 16 March 1988

202338022

TRANSACTIONS

# INDOOR AIR QUALITY IN COLD CLIMATES

## Hazards and Abatement Measures

Douglas S. Walkinshaw, Editor

An APCA Specialty Conference  
*Sponsored by*  
APCA's TT-7 Indoor Air Quality Committee  
and  
Health and Welfare Canada  
National Research Council Canada  
Public Works Canada  
Consumer and Corporate Affairs Canada  
Canada Mortgage and Housing Corporation

*Hosted by*  
Quebec Section and Ottawa Valley Chapter  
Air Pollution Control Association  
April 1985  
Ottawa, Ontario



2023380273

TR-6

**Indoor Air Quality in Cold Climates:  
Hazards and Abatement Measures**

**A Peer-Reviewed Publication**

**Copyright 1986**

**This collective work is protected by copyright. No technical paper appearing in this Transactions may be reproduced in any form without permission from the author(s) and from the publisher:**

**Air Pollution Control Association  
P. O. Box 2861, Pittsburgh, PA 15230**

2023380274

CONFERENCE COMMITTEE

GENERAL CONFERENCE CHAIRMAN

Emmanuel Somers  
Health and Welfare Canada

TECHNICAL PROGRAM CHAIRMAN

Douglas S. Walkinshaw  
National Research Council of Canada.

TECHNICAL PROGRAM CO-CHAIRMAN

James A. Frazier  
National Academy of Sciences.

TECHNICAL PROGRAM COMMITTEE

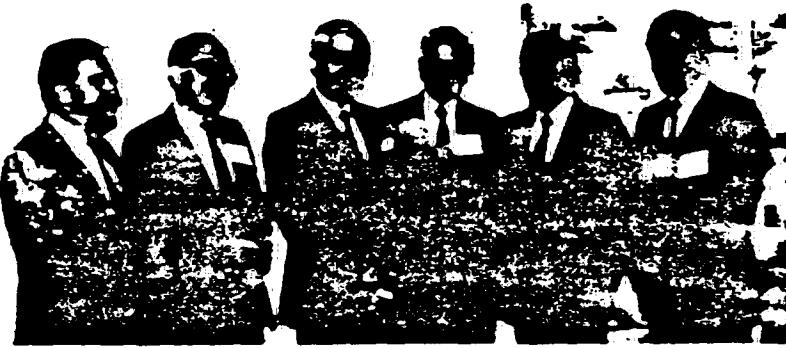
David T. Grimerud  
Gustav O. Handegord  
Israel Hoffman  
John D. Spengler  
Peter Toft  
A. Grant Wilson  
James E. Woods

CHAIRMAN, HOST PLANNING COMMITTEE

AND OTTAWA VALLEY CHAPTER  
Stanley O. Winthrop.

CHAIRMAN, QUEBEC SECTION

Robert Hudon



Show left to right: Dr. St. O. Winthrop, Chairman, Host Planning Committee and Ottawa Valley Chapter, APC; Dr. E. Somers, General Conference Chairman; Hon. Jake Epp, Minister of Health and Welfare Canada; Mr. Joseph Padgett, President, APC; Dr. Larkin Kerwin, President, National Research Council; and Dr. Douglas Walkinshaw, Technical Program Chairman and Transactions Editor.

2023380275

## PREFACE

Increasingly widespread concern has arisen during the past few years over the quality of air in residences, schools, offices, hospitals and even ice arenas as it may be affecting human health and comfort, and even the performance of materials and equipment within the building. In some cases specific indoor air pollutants have been identified as the causal agents in illness and death, as in the case of Legionnaire's disease from bacterial water droplet aerosols and carbon monoxide poisoning from combustion product venting failures. More often, however, the concern is associated with perceived but not well understood problems which have been conveniently grouped together and labelled as the 'sick building syndrome'.

With respect to the importance of the IAQ problem, at least three facts seem certain. Firstly, concerns about three specific indoor air pollutants: asbestos, passive tobacco smoke and formaldehyde, have already resulted in costly building and/or industry measures being taken. Literally hundreds of millions of dollars have been spent on asbestos and Urea Formaldehyde Foam Insulation (UFFI) removal and encapsulation. Standards have been developed in some countries for particleboard formaldehyde emissions, and significantly higher ventilation rates have been set by ASHRAE for settings where tobacco smoking is permitted.

Secondly, there are many potentially harmful gases, particulates, and microbiological agents which have been discovered in the indoor air for which no standards or policies exist. So many in fact that authorities are uncertain where to focus their IAQ efforts or even how to manage them. The problem is exacerbated by the fact that no one federal agency seems to have both the health and building mandates required to deal with the breadth of the problem holistically.

Finally, indoor air quality concerns in cold climates have been closely linked to the energy conservation measures introduced in the last decade, particularly those reducing indoor-outdoor air exchange since this act directly raises the concentrations of the many pollutants originating indoors. Therefore, a third measure of the importance of the indoor air quality concern is the importance attached to the efficiency of energy use in buildings. In Canada, at least, this turns out to be very significant. Building energy conservation measures are currently saving Canadians an estimated \$5 billions annually in comparison to their pre-1973 energy usage rates. Ventilation reductions comprise a significant but unknown amount of that \$5 billion. The challenge facing people living in cold climates is to maintain and even increase these building energy savings while minimizing indoor air pollutant concentrations so that they can live in healthful and comfortable, yet economical, indoor environments.

2023380276

It seems clear, therefore, that potential IAQ impacts must be given increased attention, particularly in cold climate areas where people spend so much time indoors. This attention is required when designing and operating combustion devices, air handling systems and humidifiers, when defining the range of acceptable indoor human activities such as tobacco smoking, and when selecting and maintaining building materials and furnishings. Action will have to be taken on the basis of available information on pollutant dose-human response, and societal cost-benefit and relative risk trade-offs.

This first APCA Specialty Conference on indoor air quality, held on April 29, 30 and May 1, 1985 in Ottawa, Canada, featured some 67 presentations covering many aspects of these issues, with the focus on cold climate hazards and abatement measures. The attendance of almost 400 persons from a variety of building, health and environmental disciplines and interests was indicative of the great interest in this topic. The conference transactions contain 38 peer-reviewed research papers, 17 policy and practice papers, and the opening addresses of the Canadian Minister of Health and the President of the National Research Council of Canada. The combined efforts of the organizers, authors and reviewers have resulted in a document which should be of use to all of those involved or interested in the field of indoor air quality.

January 6, 1986

Douglas S. Walkinshaw

2023380277

CONTENTS

OPENING ADDRESSES

- |  |   |
|--|---|
| 1. The Honourable Jake Epp, Minister of Health and Welfare Canada    | 1 |
| 2. Dr. Larkin Kerwin, President, National Research Council of Canada | 4 |

HAZARDS AND POLLUTANT SOURCES RESEARCH

- |   |     |
|---|-----|
| 3. Building Illness in a Large Office Complex<br>J.C. McDonald, M. Arhiria, B. Armstrong, J. Béhard, M.M. Cherry, D. Cyr,<br>J.P. Parent, T. McKenna and D. McKinnon .....  | 7   |
| 4. Relating Health and Environmental Studies in a Tight Building Syndrome<br>Investigation<br>J.M. Béhard, T.A. McKenna, and D.L. McKinnon .....  | 23  |
| 5. A Method for the Objective Evaluation of Indoor Air Quality Using a<br>Passive Monitoring System Coupled to Biosassays<br>M.R. Samoiloff, O.N. Bhatnagar and H.D. Gesser .....                                 | 35  |
| 6. Pulmonary Function Responses to Passive Cigarette Smoke Exposure<br>R.B. Urch, P. Silverman, P. Corey and R.J. Shephard .....  | 48  |
| 7. The Effect of the Return of Exhausted Building Air on Indoor Air Quality<br>D.D. Reible, P. Younts and P.H. Shair .....  | 62  |
| 8. The Relationship Between Indoor Concentrations of Ionic Substances and<br>Their Accumulation on Surfaces<br>J.D. Sinclair, L.A. Peota-Kelty, C.J. Weschler and S.P. Kelty .....                                | 72  |
| 9. NO <sub>2</sub> Concentrations in Offices with Kerosene Space Heaters and Electric<br>Stoves<br>Y.S. Kim, J.D. Spengler and Y. Yanagisawa .....  | 83  |
| 10. The Effect of Mechanical Ventilation on Rn, NO <sub>2</sub> and CH <sub>2</sub> O Concentrations<br>in Low-Leakage Houses and a Simple Remedial Measure for Reducing Rn<br>Concentration<br>R.S. Dumont ..... | 90  |
| 11. Objectives and Results of Radon Surveys in Saskatchewan<br>R.R. Sennis and E.P. Wagner.....   | 105 |
| 12. Radiation Exposure From Airtightening of Dwellings in Alpine Areas With<br>Cold Climate and Elevated Radium Content<br>W. Burkart .....   | 116 |
| 13. Prevalence of Formaldehyde Concentrations in Residential Settings<br>S. Syrotynski .....  | 127 |
| 14. Irritancy Levels and Formaldehyde Exposure in U.S. Mobile Homes<br>S.M. Lamm .....  | 137 |
| 15. Exposure to a High Concentration of Formaldehyde - A Case Study<br>R. Marchant, K. Yoshida and D.A. Pigley .....  | 148 |

2023380278

16.	Health Status of Residents in Homes Insulated with Urea Formaldehyde Foam E. Broder, P. Corey, P. Cole, S. Mintz, M. Lips and J. Nethercott .....	155
17.	UFFI-Its Role as a Model in the Study of Indoor Air Quality Problems: J.H. Day .....	167
18.	The Nematode Bioassay for Toxicity of Extracts of Urea-Formaldehyde Foam Insulation Removed from Three Winnipeg Homes M.R. Samoiloff .....	182
19.	Seasonal Variations in Formaldehyde Concentrations in Homes Insulated With Urea-Formaldehyde Foam J.B. Sullivan, C.J. Shirtliffe, and J.M. Svec .....	191
20.	Measurement of Off-Gases from Urea Formaldehyde Foam Insulations (UFFI) Y. Tsuchiya, R. Dussault, S.J. Rolfe, T.R. Khan, and C.J. Shirtliffe ....	208
21.	Surveys of Selected Organics in Residential Air R. Olson and F.M. Benoit .....	224
22.	Volatile Organic Compounds in Indoor Air: A Survey of Various Structures J.D. Pleil, K. Oliver and W.A. McCleney .....	237
23.	The Relationship Between Pollutant Levels in Homes and Potential Sources H.R. Hosseini, P. Silverman, P. Corey and S. Mintz .....	250
24.	Nitrogen Dioxide Levels in Residences - Effects of Source Types T. Godish and I. Ritchie .....	261
25.	A Predictive Assessment of Air Pollution Exposures to Campers Using Stoves and Lanterns Inside of Tents M.A. Cohen .....	267

#### POLLUTANT ABATEMENT RESEARCH

26.	Building Assessment Techniques for Indoor Air Quality Evaluations D.W. Bearg and W.A. Turner .....	276
27.	Implications of Providing Required Outside Air Quantities in Office Buildings J.D. Cowan .....	284
28.	Transient Modelling of Indoor Air Quality G.D. McIntosh, G.M. Bragg, and D.L. Sturua .....	297
29.	Impact of a New Smoking Policy on Office Air Quality H.K. Lee, T.A. McKenna, L.N. Renton and J. Kirkbride .....	307
30.	Office Energy Conservation: The Effect on Airborne Particles and Their Chemical Constituents C.J. Weachler .....	323
31.	Ventilation Intake Air Contamination by Nearby Exhausts D.J. Wilson .....	335

2023380279

2023380280

32.	The Effects of Occupancy and Furnace Operation on Residential Indoor Air Quality D. Desrochers and A. Robertson .....	368
33.	Residential Ventilation Rates and Indoor Radon Daughter Levels D. Desrochers and A.G. Scott .....	362
34.	Detailed Field Tests of Radon Control Techniques in New York State Houses L.A. Nitschke, J.B. Wadach, W.A. Clarke, G.W. Traynor, T. Brennan, G.P. Adams, and J.E. Rizzuto .....	369
35.	Preliminary Evaluation of Formaldehyde Mitigation Studies in Unoccupied Research Homes T.G. Matthews, W.C. Dreisbach, C.V. Thompson and A.R. Hawthorne .....	389
36.	Formaldehyde Emissions From Typical Particleboard Applications and Assessment of Specific Abatement Measures R. Kalnins and P.C. Gaudet .....	402
37.	Avoidance of Chimney Backdrafting in Houses: Identifying the Critical Conditions M.C. Swinton and J.H. White .....	413
38.	Combustion Ventilation Hazards in Housing - Failure Mechanisms, Identification Technologies, and Remedial Measures S. Moffatt .....	423
39.	Burner Flame Inserts for Lower NO <sub>x</sub> Emissions D.W. DeWerth and R. Kurzynski .....	437
40.	Room Temperature Catalyst for Improved Indoor Air Quality M.P. Collins .....	448

#### POLICY AND PRACTICE

41.	Updating ASHRAE 62-1981: Problems and Issues J.E. Janssen and D.T. Grimesrud .....	461
42.	Exposure Guidelines for Residential Indoor Air Quality H.W. Wyllie and V. Armstrong .....	468
43.	Regulatory Aspects of Indoor Air Quality - A U.K. View J.W. Llewellyn and P.R. Warren .....	478
44.	A Radon Standard for Canada E.G. Létourneau .....	488
45.	The Canadian UPPI Assistance Program R.W. Shurb .....	496
46.	Passive Tobacco Smoking Policies Indoors in Various U.S. States D.R. Shopland .....	504
47.	The U.S. Indoor Air Quality Research Strategy J.P. Millhone and K.Y. Teichman .....	523

4. Improving Indoor Air Quality Through Energy Conservation Programs C. Fischer, M. Riley and M. Lubun .....	534
5. Bonneville Power Administration's Risk Management Strategy for Mitigating Indoor Air Quality Effects in its Home Weatherization Program R.L. Love .....	549
6. Industry/Government Cooperation and Coordination in Indoor Air Quality Research I.M. Billick .....	561
7. Cooperative Indoor Air Quality Research Between Government and Industry - A Minnesota Case Study C.A. Lane, J. Armstrong, J. Lind and J.E. Woods .....	574
8. Electric Power Research Institute Indoor Air Quality Research Program G.G. Purcell .....	586
9. A Community Indoor Air Pollution Program R. Marchant, B. Wensley, D. Frei, J. Bartram and K. Yoshida .....	586
10. Diagnosis and Management of Hypersensitivity Reactions to Indoor Air Pollution in the Susceptible Individual J.G. Maclellan .....	589
11. Diagnosis and Management of the Impact of Indoor Air Pollution on the Behaviour and Learning Ability of School Children J.G. Maclellan .....	599
12. Low Pollution Building Design and Construction B.M. Small .....	607
13. Appropriate Technology for Residential Air Quality K.H. Raab .....	614
ACKNOWLEDGEMENTS .....	617
REVIEWERS .....	618
INDEX .....	620

2023380281

INDOOR AIR QUALITY IN COLD CLIMATES--HAZARDS AND ABATEMENT MEASURES

ACKNOWLEDGEMENTS

The time period over which the conference, its organization and the subsequent publication preparation took place, coincided with a period when a substantial reduction in Canadian federal energy-funded indoor air quality research was occurring. It was, therefore, especially gratifying to receive such a strong international response to the Call for Papers, to have such an unexpectedly large attendance at the conference, and to have such a substantial effort by so many authors and reviewers to produce the papers finally contained in this document.

For their special efforts I particularly wish to acknowledge:

- Mr. James Frazier, U.S. National Academy of Sciences, who suggested the need for a focus on the cold climate issue.
- Mrs. Betty Gallimore, my former secretary, who managed a large conference correspondence load and still found time to produce the Abstracts booklet provided at the Conference.
- Dr. Larkin Kerwin, President of NRC, and Dr. Carl Crawford, former Director of NRC/DBR, who have provided considerable support to the NRC IAQ effort.
- Drs. E. Somers, S. Winthrop, P. Toft, J. Kirkbride and Mt. R. Hickman of Health and Welfare Canada for their support in all aspects of this event.
- The Honourable Jake Epp, Minister of Health and Welfare Canada, for making such an eloquent plea during his presentation for the elimination of the involuntary tobacco smoke hazard to non-smokers.
- Members of the Conference Committee who spent many hours, together and on Conference Calls, to produce this event.
- Messrs. Steve Hart, John McGovern and Hal Englund of APCA for their interest and knowledgeable advice throughout.
- Dr. Al Hoffman, Mr. Grant Wilson and Mr. Clint Mitaless for their provision of wise advice whenever called upon.
- And, most importantly, my wife, Barbara, and children, Chris and Caroline, for their interest in my work and support for my concerns.

2023380282

THE RELATIONSHIP BETWEEN POLLUTANT LEVELS  
IN HOMES AND POTENTIAL SOURCES

Roland Hosein,

Frances Silverman, Paul Corey, and Shelley Mintz  
Occupational Health Unit, Faculty of Medicine,  
and the Gage Research Institute  
University of Toronto, Toronto, Ontario, Canada, M5S 1A8



Indoor levels of respirable suspended particulates (RSP), nitrogen dioxide ( $\text{NO}_2$ ) and sulphur dioxide ( $\text{SO}_2$ ) were measured in homes in two health related studies. The air pollution sampling was conducted using a specially designed system which sampled the pollutants simultaneously.

The potential sources of the air pollution investigated included heating systems, cooking fuel, air conditioners, number of occupants, pets, furniture, floor coverings, tobacco smoke, and fireplaces.

Study I showed that  $\text{NO}_2$  levels were higher in homes with gas stoves than in homes with electric stoves. The difference averaged 100  $\mu\text{g}/\text{m}^3$  with a maximum level detected of 3,000  $\mu\text{g}/\text{m}^3$ . The levels of RSP in homes was directly related to the number of smokers. Homes with one and two smokers had RSP levels of 14  $\mu\text{g}/\text{m}^3$  and 39  $\mu\text{g}/\text{m}^3$  respectively higher than homes with no smokers.  $\text{SO}_2$  levels were very low and there were no obvious indoor sources of this pollutant.

Study II investigated  $\text{NO}_2$ , (suspended particulate matter), and  $\text{SO}_2$ . Homes with gas stoves were excluded from this study. Homes with fireplaces had 18  $\mu\text{g}/\text{m}^3$  more  $\text{NO}_2$  and 6  $\mu\text{g}/\text{m}^3$  less RSP than homes without fireplaces;  $\text{SO}_2$  levels were very low and similar in both types of homes.

Air conditioning usage was found to influence the  $\text{NO}_2$  and RSP levels to a small extent. The other sources had very little influence. In general, indoor  $\text{NO}_2$  and RSP levels were higher than outdoor levels.

2023380283

## INTRODUCTION

It is now recognized that the indoor environment is important in an individual's total exposure to airborne contaminants since in some areas residents spend 70 - 90% of their time indoors<sup>1,2</sup>. The indoor home environment has numerous sources of airborne respiratory irritants. Smoking, cooking, cleaning, heating and fireplace use all contribute to the generation of indoor airborne pollution<sup>2,3,4</sup>.

In general, indoor SO<sub>2</sub> concentrations are usually less than outdoors unless there are particular indoor sources<sup>5</sup>. Indoor NO<sub>2</sub> levels can be higher depending on indoor sources<sup>6</sup>. A significant indoor source of NO<sub>2</sub> has been shown to be gas stoves<sup>7,8,9,10</sup>. Tobacco smoke is a major source of indoor airborne particulate matter and to a lesser extent NO<sub>2</sub><sup>11</sup>. Airborne sulfonate levels have been shown to be associated with smoking and use of matches<sup>12</sup>.

Two studies were conducted to determine the effects of indoor pollution on the respiratory health of residents. In study I, a case/control study was conducted with a group of housewives in which the cases reported chronic cough and phlegm. In study II, another case/control study was conducted in which the cases reported asthma.

In both studies lifestyle factors were evaluated to determine the contribution to airborne SO<sub>2</sub>, NO<sub>2</sub> and RSP, which were measured simultaneously using uniquely designed samplers.

## METHODS

### Study I

The air pollution sampler consisted of a suitcase (50 x 88 x 15cm). A manifold connected to a blower served as the main sampling system from which probes lead to 3 pumps. One of the pumps sampled respirable suspended particulates on to glass fiber filters using a 10mm nylon cyclone assembly. Two other pumps collected NO<sub>2</sub> and SO<sub>2</sub> by bubbling air into iningers with sodium hydroxide and potassium tetrachloromercurate solutions respectively. The level of NO<sub>2</sub> was determined using the Jacobs and Hochneiser method<sup>13</sup> whereas the SO<sub>2</sub> was determined by the West and Gaede method<sup>14</sup>. RSP was determined by the gravimetric method.

The sampler was placed in the area most frequently used by the subject. Samples were collected once in the summer and winter for each subject. The sampling period was 24 hours.

2023380284

2023380285

The lifestyle characteristics considered were: air conditioning, cooking fuel, smoking, type of heating system, and carpets on the floor.

#### Study II

The air pollution sampler was a variation of the one used in Study I. It was made more compact to be used as a personal or indoor sampler. It collected the same gases, but the particulate matter consisted of particles less than 25  $\mu\text{m}$  (SPM). Samples were collected everyday for two weeks in the heating and two weeks in the non-heating seasons. The samplers were operated for 6 to 8 hours, primarily during the day time. Details of the sampler design were given elsewhere<sup>15</sup>.

The lifestyle factors included: heating system, air conditioning, smoking, floor covering, wall covering, fireplace, air tightness of home and crowding.

The data for each individual were averaged and a t-test was conducted to test the differences between the means for those who had or did not have the lifestyle characteristic.

In both studies, comparison measurements were undertaken outside of the homes using similar sampling and analytical methods.

#### RESULTS

##### Study I

In order to determine whether there were any indoor sources of the 3 pollutants, indoor levels were compared with outdoor levels. The data show that there were indoor sources of  $\text{NO}_2$  and RSP but not  $\text{SO}_2$ . In fact the  $\text{SO}_2$  levels were very low both indoors and outdoors, and no further analysis of the  $\text{SO}_2$  results are given (Figure 1).

##### Sources of $\text{NO}_2$

Twenty-one percent of the homes had gas stoves, and in these homes the nitrogen dioxide levels (Figure 2) were significantly higher than homes with electric stoves ( $P < .001$ ). This differential between homes with gas and electric stoves was seen in both seasons. The winter  $\text{NO}_2$  levels were higher than the summer in homes with both gas and electric stoves. Homes with gas stoves and air conditioning had less  $\text{NO}_2$  than homes without air conditioning, but the numbers in each group were too small for meaningful comparisons (Table I).

Five percent of the homes had gas radiant heaters, but some of these homes also had gas stoves, hence it was not possible to assess the emissions of NO<sub>2</sub> from this source. The homes of smokers without gas stoves had more NO<sub>2</sub> than homes of non-smokers without gas stoves. Although the differences were significant ( $P < .001$  and  $P < .02$  respectively), the absolute differences in the geometric means were small (Table II).

The tests conducted to determine the daily variations in domestic NO<sub>2</sub> loading showed that in two homes with gas ranges, the NO<sub>2</sub> levels were sometimes in excess of 1000 ug/m<sup>3</sup> (i.e. 2-hourly values). In one home, thirteen of the sixty 2-hourly values were in excess of 1000 ug/m<sup>3</sup> and in the other home, eight of the sixty 2-hourly values were in excess of 1000 ug/m<sup>3</sup>. In this latter home, peak 2-hourly values in excess of 3000 ug/m<sup>3</sup> were observed on two separate occasions. In all cases, these peaks were directly related to extensive use of the gas stoves and ovens.

#### Sources of RSP

Cigarette smoking contributed significantly to the RSP loading within homes in the winter and summer (Figure 3). The presence of one smoker in the home resulted in significantly more RSP than homes with no smokers ( $P < .001$ ), and homes with 2 or more smokers showed more RSP than homes with one smoker or no smokers ( $P < .001$ ). In homes with air conditioning, the RSP levels were higher in both homes with zero smokers ( $P < .01$ ) and with at least one smoker ( $P < .001$ ) compared with homes without air conditioning (Table III).

Carpeted houses with at least one smoker had more RSP than non-carpeted houses with at least one smoker (Table IV), but houses of non-smokers with carpet had less RSP than houses of non-smokers without carpets.

Homes with hot water heating with no smokers had significantly higher levels of RSP than homes heated by forced air. Homes with one or more smokers and with hot water heating had significantly higher levels of RSP than homes heated by forced air (Table V).

#### Study II

Indoor NO<sub>2</sub> concentrations were higher in homes with at least one fireplace than in homes without any fireplace (Heating Season only) (Table VI). There was no effect of fireplaces on the indoor levels of SO<sub>2</sub>. The indoor SPM concentrations were higher in homes without fireplaces than in homes with fireplaces.

2023380286

2023380287

The presence of smokers in the home had no effect on the concentrations of SO<sub>2</sub> and an inconsistent effect on NO<sub>2</sub> levels. Indoor SPM levels were higher in homes with smokers.

The use of gas heating on the indoor levels showed that there were higher indoor NO<sub>2</sub> levels (Heating Season only) but lower SPM levels (Table VI) compared to homes with other types of heating.

Homes reported as being airtight did not differ in their indoor SO<sub>2</sub> levels from homes that were reported as permeable. Any differences observed for indoor concentrations of NO<sub>2</sub> and SPM were inconsistent (Table VI).

The indoor concentration of NO<sub>2</sub> tended to be higher in homes with more than two adults (Table VI). Very little difference was observed for SO<sub>2</sub>. The observed differences in the concentration of SPM were small and inconsistent. The indoor concentrations of NO<sub>2</sub> were higher in homes with no children (Table IV). No differences were observed for SO<sub>2</sub> and any differences observed for SPM were inconsistent.

The indoor levels of NO<sub>2</sub> were lower for homes that had pets that would go in and out of doors (Table VI). Pets had no effect on SO<sub>2</sub> or SPM.

Homes with air-conditioners (Table VI) had lower NO<sub>2</sub> and SPM than homes without air-conditioners. In this sample only asthmatics had air cleaners (Table VI). The indoor concentrations of SPM were higher in home without air cleaners.

Homes with carpets had more SPM than homes without; there were no effects of carpets on SO<sub>2</sub> and NO<sub>2</sub> levels. Homes with wallpaper had lower SPM than homes without wallpaper; there were no effects of wallpaper on SO<sub>2</sub> and NO<sub>2</sub> levels. Homes with soft furniture had lower NO<sub>2</sub> and higher SPM than homes without soft furniture.

#### CONCLUSION

The levels of SO<sub>2</sub> were low both inside and outside, and the outdoor levels were generally higher than the indoor levels. With regards to NO<sub>2</sub>, gas stoves were the major contributors to the high levels measured whereas smoking indoors was a very small contributor to NO<sub>2</sub>.

Smoking accounted for the high indoor levels of particulates, and carpeted homes seemed to have higher levels of suspended particulates.

Crowding, air tightness and pets had no consistent effects on the indoor pollutant levels.

REFERENCES

1. A. Szalai. *The use of time*. Mouton, Paris, 1972.
2. R. Binder, C. Mitchell, H.R. Mosein, J. Bourdy, "Importance of the indoor environment in air pollution exposure," *Atmos. Environ. Health*, 31:277 (1976).
3. J.E. Vocom, A.L. Cline, W.A. Cote, "Indoor-outdoor air quality relationships," *J. Air Pollut. Contr. Assoc.*, 21(5): 251 (1971).
4. V. Lefebvre, I. Inculet, "Particulates in domestic premises," *Atmos. Environ. Health*, 30: 565 (1975).
5. K. Biersteker, M. deGraaf, C. Pimattio, "Indoor air pollution in Rotterdam homes", *Int. J. Air Water Pollut.*, 9:243 (1965).
6. R.J.W. Melia, C du V Florey, S.C. Darby, E.O. Barnes, B.C. Goldstein, "Differences in NO<sub>2</sub> levels in kitchens with gas or electric cookers," *Atmos. Environ.*, 12: 1379 (1978).
7. B.C. Goldstein, R.J.W. Melia, S. Chinn, C du V Florey et al., "Factors affecting NO<sub>2</sub> levels in the home," *Int. J. Environ.*: 339 (1979).
8. F.E. Speizer, B. Ferris, V.M.M. Bishop, J. Spengler, "Respiratory disease rates and pulmonary function in children associated with NO<sub>2</sub>," *Am Rev. Resp. Dis.*, 121:3 (1980).
9. H. Marke, "The influence of tobacco smoke on indoor atmospheres," *Prev. Med.*, 4: 373 (1976).
10. G. moek, R. Meijer, A. Schollen, D. Noij, E. Lebret, "The relationship between indoor NO<sub>2</sub> concentration and personal exposure," *Int. Arch. Occup. Environ. Health*, 55: 73 (1984).
11. A. Weber, T. Fischer, "Passive smoking at work", *Int. Arch. Occup. Environ. Health*, 47:209 (1980).
12. D. Dockery, J.D. Spengler, "Personal exposure to respirable particulates and sulfates," *J. Air Pollut. Contr. Assoc.*, 31: 153 (1981).
13. W.B. Jacobs and S. Hochreiser, "Continuous sampling and ultra-microdetermination of NO<sub>2</sub> in air". *Anal. Chem.* 30:426 (1958).
14. P.W. West and G.C. Gaede, "Fixation of SO<sub>2</sub> as sulfite-mercurate III and subsequent colorimetric determination." *Anal. Chem.* 28:1816 (1956).
15. S. Mintz, H.R. Mosein, B. Batten, F. Silverman, "A personal sampler for 3 respiratory irritants". *J. Air Poll. Contr. Assoc.*, 32:1068 (1982).

2023380288

TABLE I  
Nitrogen Dioxide Levels in Homes with Gas and Electric Stoves  
with and without Air Conditioning  
(Summer Levels Only)

	<u>With Air Conditioning</u>		<u>Without Air Conditioning</u>	
	<u>Electric</u>	<u>Gas</u>	<u>Electric</u>	<u>Gas</u>
No. of obs.	12	4	29	7
Geometric Mean (ug/m <sup>3</sup> )	70.9	175.1	81.8	192.1
Geometric Standard Deviation	2.00	1.56	1.70	1.62

TABLE II  
Nitrogen Dioxide Levels in Homes without Gas Stoves  
as a Function of Smoking

	<u>Winter</u>		<u>Summer</u>	
	<u>Smoker</u>	<u>No Smoker</u>	<u>Smoker</u>	<u>No Smoker</u>
No. of obs.	29	12	29	12
Geometric Mean (ug/m <sup>3</sup> )	82.6	75.7	76.1	74.5
Geometric Standard Deviation	1.75	1.23	1.74	1.93
Unpaired t-test:	P<.001		P<.02	

2023380289

TABLE III

Respirable Suspended Particulates: Exposure of Smokers and Non-Smokers as a Function of Air Conditioning (AC)

(Summer Levels Only)

	<u>Zero Smokers in home</u>		<u>At least one Smoker in home</u>	
	<u>AC</u>	<u>No AC</u>	<u>AC</u>	<u>No AC</u>
No. of obs.	4	11	11	25
Geometric Mean (ug/m <sup>3</sup> )	34.3	32.5	80.5	70.11
Geometric Standard Deviation	1.60	1.64	1.40	1.64
Unpaired t-test:		P<.01		P<.001

TABLE IV

Respirable Suspended Particulates: Exposure of Smokers in Houses with and without Carpets

	<u>Smoker Home</u>		<u>Non-Smoker Home</u>	
	<u>Carpeted</u>	<u>Non-Carpeted</u>	<u>Carpeted</u>	<u>Non-Carpeted</u>
No. obs.	28	8	9	7
Geometric Mean (ug/m <sup>3</sup> )	76.6	70.2	38.7	53.7
Geometric Standard Deviation	1.68	2.14	1.91	1.73
Unpaired t-test:		P<.001		P<.001

2023380290

TABLE V  
RSP as a Function of Smoking and Heating

	Homes			
	Smoker		Non Smokers	
	Hot Water	Forced Air	Hot Water	Forced Air
Geometric Mean (ug/m <sup>3</sup> )	84.8	57.1	66.7	37.7

TABLE VI  
Pollutant Concentration as a Function of Sources

POLLUTANT SOURCE	Concentration (ug/m <sup>3</sup> )					
	NO <sub>2</sub>		SO <sub>2</sub>		SPM	
YES	NO	YES	NO	YES	NO	
* Fireplace	24	17	3	3	61	70
* Smokers in Home	25	27	3	3	83	63
* Gas Heating	23	18	2	3	55	77
Air Tight	26	26	6	2	71	70
At least one child	23	31	3	3	71	69
More than 2 adults	31	24	3	2	71	70
Pets	25	29	3	3	71	69
** Air Conditioning	35	40	2	2	75	79
** Air Cleaner	27	26	1	3	65	70
Carpet	26	26	3	3	73	66
Wallpaper	27	26	3	4	66	73
Soft Furniture	21	30	4	2	83	65
* Heating Season data only						
** Non-heating season data						
*** Asthmatic only						

2023380291

2023380292

FIGURE 1

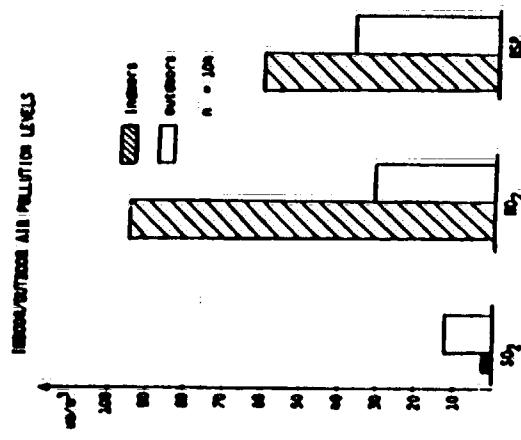
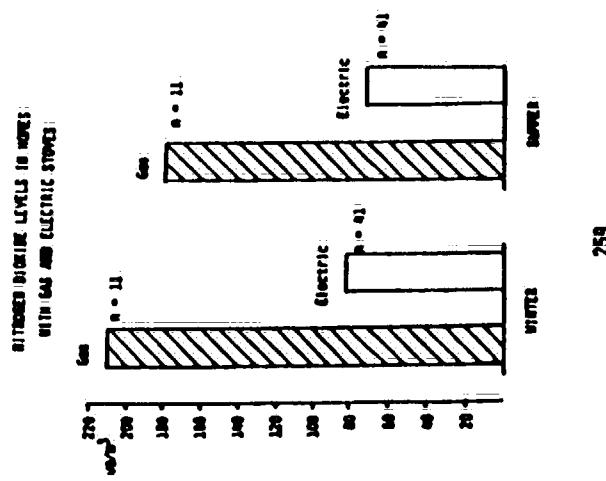


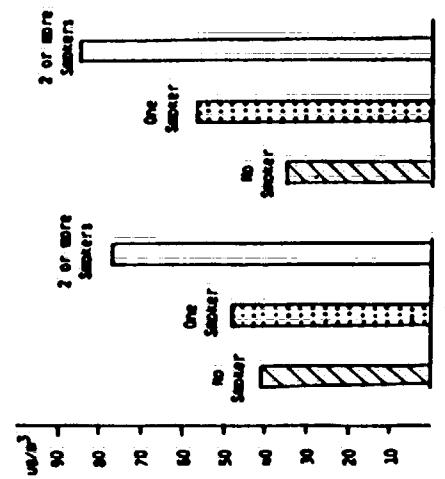
FIGURE 2



259

2023380293

FIGURE 3  
RESPIRABLE SUSPENDED PARTICULATE LEVELS  
AS A FUNCTION OF SPKIES



260

## MEASUREMENTS OF ENVIRONMENTAL TOBACCO SMOKE IN AN AIR-CONDITIONED OFFICE BUILDING

C. J. Proctor\*, N. D. Warren and M. A. J. Bevan

BAT(UK & E) Research and Development Centre,  
Regents Park Road, Southampton, SO9 1PE, U.K.

(Received 12 May 1989; in final form 18 September 1989)

*NOTICE*  
This material may be  
protected by copyright  
law (Title 17 U.S. Code).

### ABSTRACT

This paper reports levels of nicotine, respirable particulates, carbon monoxide, carbon dioxide and volatile organic compounds measured in the air of smokers' and non-smokers' offices in a modern air-conditioned building. The results show very low levels of environmental tobacco smoke constituents, such as nicotine, present in smokers' offices. Moreover, the data show that smoking has little influence on the levels of volatile organic compounds found in the office air.

### INTRODUCTION

Environmental Tobacco Smoke, ETS, is the complex mixture of chemicals found in air as a specific result of smoking (1). Some reports have claimed that ETS is harmful to the health of the non-smoker (2,3,4). This issue has been discussed by scientists and doctors for over a decade, and although knowledge has increased over this period, it is still the subject of scientific controversy (2, 5). The claims have primarily been based on combining the results of epidemiological studies that have all been stated to be, when taken individually, inadequate (2, 3, 4, 6). Several experts in the field of low-risk epidemiology have stated that it is not possible to draw firm conclusions as to whether or not ETS is harmful to the health of the non-smoker (5, 6, 7).

In spite of the continuing debate, there have been calls for the introduction of further restrictions on where smoking can take place (4, 8). Much attention has recently focused on the work place, and in particular to the modern office environment.

Ever since the energy crisis of the 1970's, many of the office buildings constructed in the Western world have been designed with air conditioning systems that limit energy costs and assist in energy conservation. Often control over the amount of fresh air taken into the building is determined simply by the temperature measured at various points.

It has been well documented that buildings release chemicals into the air (9). Building materials, furnishings and coverings, and the building occupants will all contribute to the chemical burden of the office air (10). In extreme cases this may result in 'Sick Building Syndrome', where the occupants suffer symptoms and discomforts including headaches, burning eyes, irritation of the respiratory system, drowsiness, fatigue and general malaise (11). The causes of Sick Building Syndrome are

2023380294

not entirely defined, but are likely to be primarily exposure to bacteria, moulds and fungi produced and circulated by poorly maintained ventilation systems, and exposure to volatile organic compounds produced by various sources (12, 13).

The United States Environmental Protection Agency claimed, as part of the conclusions to their Total Exposure Assessment Methodology (TEAM) Studies, that the presence of ETS results in significantly higher levels of volatile organic compounds in air (14). Other authors, using similar methodologies, were unable to distinguish between smoking and non-smoking environments when measuring ambient volatile organics (15).

This paper presents the results from an investigation of the air in offices in a modern air-conditioned building located in Southern England. Ten offices, of different sizes, population and density of smokers, were each visited on five separate occasions. On each occasion, measurements were made of levels of nicotine, respirable suspended particulates, carbon monoxide, carbon dioxide and volatile organic compounds in the air.

### THE BUILDING

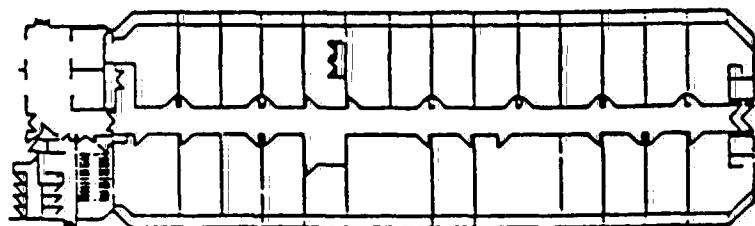
The building selected for this study is a 1970's built office block comprising of around 9300 square metres of floor space on 16 floors, and holding around 350 people. Although originally designed to be open plan, it has been modified over the years to incorporate a modular office design, though some open plan areas remain.

Air conditioning is nominally the same in all areas. There are two systems, one at the perimeter and one operating through the core of the building. Air for both systems is drawn from the roof where it is filtered and humidified. The perimeter system enters on each floor at vents positioned on window sills and exits through vents in the ceiling at the centre of the building. This system is monitored for temperature and relative humidity on three floors (floors 14, 11 and 5), conditions being fed-back to a central controlling system. The volumetric flow rate for the perimeter system was  $17.5 \text{ m}^3 \text{ s}^{-1}$ . With 544 vent outlets, the volumetric flow per module was  $1.93 \text{ m}^3 \text{ min}^{-1}$ , resulting in a typical air exchange rate for each office of around 3 air changes per hour.

The core system operates through the centre of each floor, often in a corridor, at a total volumetric flow rate of  $17 \text{ m}^3 \text{ s}^{-1}$  and is controlled by rheostats and motorised valves on every floor. A typical floor plan is given as Figure 1.

FIGURE 1

TYPICAL FLOOR PLAN OF THE BUILDING UNDER INVESTIGATION



Maximum possible recirculation is 84% (i.e. 16% incorporation of fresh air), though this condition is rarely used and recirculation rates vary throughout the day. The entire system is operated in a manner that minimises total energy costs.

### SAMPLING SITES

10 offices were selected to represent the variety of environments within the building. This included open plan space; single and multiple occupier offices, with different populations and numbers of smokers, as detailed in Table 1.

TABLE 1: Details of Sampling Sites

Site Number	Floor	Number of Occupants		Approximate Size (m <sup>3</sup> )
		Non-smokers	Smokers	
1	12	0	1	30
2	11	0	1	60
3	10	3	1	115
4	9	3	0	80
5	7	29	1	760
6	6	2	3	180
7	5	4	1	155
8	4	3	3	90
9	4	1	0	30
10	11	1	0	60

Samples were acquired between 0900 and 1600 hrs. Each site was visited 5 times, each occasion for a particular office being at a different time of the day and on different days to avoid any bias from possible temporal variations. So, for example, site number 7 was sampled between 14.40 and 15.40 on day 3, 10.20 and 11.20 on day 5, etc.

Each sample was acquired for one hour, and the sample was taken as near as possible to the centre of the office and at approximately head height of a seated person.

No smoker segregation is imposed in this building, and so smokers are free to visit and smoke in the offices of non-smokers. As a general rule, this rarely occurred in the sites investigated in this study.

### Analytical Considerations

All of the analytical methods for the analysis of the components under investigation have been previously detailed in the scientific literature. Briefly, the methods were;

- a) Nicotine: Airborne nicotine was collected by drawing, at a rate of 1 litre per minute, air through a sorbent sampling tube containing XAD-4 resin (20/40 mesh) (SKC, Inc.) (16, 17). After sampling, the tube was capped and returned to the laboratory. The collected nicotine was extracted from the resin using a quantity of ethyl acetate, modified with 0.01% triethylamine (to prevent losses of nicotine to the glassware). Analysis was effected by capillary gas chromatography with nitrogen-phosphorous detection. With this flow rate, and sampling periods of one hour, detection limits equate to approximately 0.1 µg m<sup>-3</sup> nicotine.

2023380296

- b) Respirable suspended particulates (RSP): Airborne particulates were measured by gravimetric analysis. Air from the environment was drawn at 2 litres per minute through a fluoropore membrane filter (17) (Millipore UK Ltd) via an impactor separating at 3.5 microns. The filter was weighed on an electronic balance capable of resolving  $\leq 0.1 \mu\text{g}$  (Perkin-Elmer), before and after sampling, each time being conditioned first at 50% relative humidity, to arrive at the RSP measurement.
- c) Ultra-Violet Respirable Suspended Particulates (UV-RSP): In order to estimate the contribution of ETS to the total respirable particulates, each filter, after being weighed, was extracted with methanol and the resulting solution analysed for its absorbance at 325 nm. This was achieved by injecting the solution through a columnless liquid chromatography system into a UV detector and integrating the resultant peak. Previous research has shown that if only ETS is present (i.e. in controlled conditions) then the calculated UV-RSP value is equivalent to RSP when using 1,1,2,2-tetrahydroxybenzophenone as a surrogate for calibration (18). In real-world environments, UV-RSP will give an over-estimate of the ETS contribution, as other sources may contribute chemicals collected that also absorb at 325 nm (19).
- d) Carbon monoxide (CO): The CO monitoring system consists of a constant flow sampling pump and a detector based on electrochemical measurements (supplied by Neotonics, Gainesville, GA). Output from the detector was fed into a data logger (Campbell Scientific, Utah) which recorded signals every 30 seconds. Unlike all of the other measurements, CO analysis gave continuous real-time readings. Data given for each sample are the arithmetic mean of the readings over the sampling period.
- e) Carbon dioxide ( $\text{CO}_2$ ): Dräger tubes ( $\text{CO}_2$ , 0.01%/a, CH30 801) were used to measure ambient  $\text{CO}_2$  levels (Drägerwerk, West Germany). This measurement was made approximately 5 minutes prior to the end of each sampling period.
- f) Volatile organic compounds (VOC): Volatile chemicals present in the air were collected by drawing the air, using a fixed diaphragm pump at a rate of  $10 \text{ cm}^3$  per minute, through a stainless steel tube containing the absorbent Tenax TA (60 - 80 mesh) (20). After collection, each tube was capped and returned to the laboratory. Analysis of each sample required thermal desorption (using a Perkin-Elmer Ltd. ATD-50) for 20 minutes at  $240^\circ\text{C}$ , during which time the eluted chemicals were swept from the sampling tube to a cryofocusing trap maintained at  $-30^\circ\text{C}$  and containing a small quantity of Tenax. After this primary desorption the cold trap was rapidly heated electronically to  $240^\circ\text{C}$  whereby the chemicals were effectively injected in a narrow band onto a capillary gas chromatography column connected to an ion trap detector (a bench-top mass spectrometer supplied by Perkin-Elmer Ltd.). The capillary column (30m,  $0.32 \mu\text{m}$  ID, DB-5) separated the individual components, and the mass spectrometer both identified and quantified. For each chromatographic peak, compound identification was confirmed by its mass spectrum, and quantification used the base peak of the mass spectrum (e.g. benzene was quantified on the signal due to the m/z 78 ion). Calibration of this system required introduction of mixed standards of the compounds of interest injected at five different levels, each level in duplicate, onto a clean Tenax tube in order to run standards through the entire procedure. In order to check the performance of the instrument, a quantity of 2-bromonaphthalene (in methanol) was injected onto each tube prior to sampling. During sampling, the methanol eluted through the Tenax, but the 2-bromonaphthalene remained trapped for subsequent thermal desorption.

#### RESULTS AND DISCUSSION

The data for all smokers' and non-smokers' offices are given in Table 2. Arithmetic means, medians, minimum and maximum values are given for each of the analytes. All data points from the total of 50 visits are included. Arithmetic means are generally of a higher value than medians due to a skewed distribution of values.

TABLE 2

## SUMMARY OF DATA FROM ALL SAMPLING SITES

(Data in  $\mu\text{g m}^{-3}$  apart from CO and  $\text{CO}_2$  which is given in ppm)

	Site 1	Site 2	Site 3	Site 4	Site 5	Site 6	Site 7	Site 8	Site 9	Site 10
No. Smokers $\times 10^3 \text{ m}^{-3}$	33	17	8.7	0	1.3	17	6.5	33	0	0
NICOTINE	mean median range	8 4.7 2.19	18.1 18.1 10.26	3.6 4.1 1.5.5	0.2 0.1 0.1-0.5	1.2 1.0 0.6-2.1	6.9 6.0 4.2-11.4	2.1 2.2 1.2-3.1	2.5 2.4 0.7-4.7	0.5 0.7 0.1-0.8
RESPIRABLE SUSPENDED PARTICULATES	mean median range	97 82 41-167	148 128 91-225	91 71 40-172	116 71 69-208	97 129 19-150	109 70 43-210	80 81 49-118	101 80 67-199	52 43 27.91
UV-RSP	mean median range	23 17 1.75	61 69 29-72	7 5 1-17	11 9 2-15	8 7 2-14	30 27 21-48	13 13 9-19	18 19 4-28	3 3 1-6
CARBON MONOXIDE	mean median range	1.2 1.0 0.9-1.4	1.4 1.4 1.0-2.0	0.9 1.0 0.5-1.2	1.4 1.0 0.7-3.6	2.3 1.6 1.0-4.8	1.8 1.2 1.0-2.6	1.2 0.9 0.8-2.3	1.0 1.0 0.6-1.3	1.0 1.0 0.7-2.2
CARBON DIOXIDE	mean median range	730 800 450-1000	570 600 500-650	520 500 450-600	610 600 500-800	560 500 500-700	600 600 550-700	610 600 600-650	540 600 450-600	520 500 500-550
BENZENE	mean median range	10 10 5-14	8 8 5-13	7 6 3-14	12 9 3-23	21 13 6-49	19 13 6-48	15 7 5-46	9 5 5-21	15 12 9-31
CHLOROBENZENE	mean median range	0.2 0.2 0.1-0.4	0.2 0.2 0.1-0.4	0.2 0.2 0.1-0.5	0.3 0.3 0.1-0.4	0.6 0.8 0.1-1.0	0.4 0.3 0.1-1.0	0.4 0.3 0.1-1.1	0.3 0.3 0.1-0.6	0.7 0.4 0.3-2

1007

2023380298

TABLE 2 CONTINUED

1008

		Site 1	Site 2	Site 3	Site 4	Site 5	Site 6	Site 7	Site 8	Site 9	Site 10
N-DECANE	mean	6	8	8	5	9	12	7	6	10	4
	median	4	6	5	5	7	15	5	3	8	4
	range	4-13	1-18	1-24	1-7	2-22	2-20	2-16	3-13	6-16	1-6
1,2-DICHLORO-BENZENE	mean	0.3	0.4	0.4	0.7	0.7	0.5	0.4	0.4	0.6	0.7
	median	0.2	0.2	0.3	0.7	0.7	0.3	0.4	0.4	0.4	0.6
	range	0.2-0.6	0.1-0.9	0.2-0.8	0.2-1.1	0.2-1.4	0.1-1.1	0.1-0.7	0.2-0.5	0.2-1	0.4-1.0
1,2-DICHLORO-ETHANE	mean	11	9	13	12	14	19	16	8	17	15
	median	10	8	6	12	17	19	18	9	19	15
	range	6-16	6-13	4-37	5-18	5-24	5-39	4-21	3-12	10-27	5-19
DODECANE	mean	3	2	1	1	1	2	2	2	2	3
	median	2	2	1	1	1	2	1	1	2	1
	range	2-4	1-2	1-2	0.2-2	0.7-2	0.6-3	0.9-3	1-3	1-3	1-11
ETHYL-BENZENE	mean	5	4	3	5	11	54	6	4	5	5
	median	5	3	3	4	6	52	4	5	3	5
	range	3-6	3-8	2-4	1-13	3-26	11-122	3-15	3-5	3-13	4-7
LIMONENE	mean	7	4	3	2	5	10	7	3	4	2
	median	6	4	2	2	6	5	7	3	4	2
	range	4-11	2-8	2-8	0.4-4	1-9	4-31	5-8	2-6	2-8	1-3
N-OCTANE	mean	2	2	2	2	5	312	5	4	4	6
	median	2	2	2	2	3	485	2	5	4	2
	range	2-3	1-2	1-3	1-3	2-10	22-528	2-9	2-5	2-6	1-15
$\alpha$ -PINENE	mean	2	3	2	5	6	5	4	3	4	5
	median	2	2	2	4	6	3	4	3	3	4
	range	1-4	1-7	1-4	2-8	2-11	3-11	2-7	2-5	3-7	2-7
STYRENE	mean	6	9	7	11	27	17	19	10	26	16
	median	5	8	4	12	42	6	13	10	15	15
	range	2-16	4-17	3-21	6-16	4-50	4-53	3-59	3-20	7-79	4-28

2023380299

TABLE 2 CONTINUED

	Site 1	Site 2	Site 3	Site 4	Site 5	Site 6	Site 7	Site 8	Site 9	Site 10
TETRACHLOR-ETHENE	mean median range	2 2 1.3	3 3 1.6	2 2 1.4	3 3 1.8	3 2 1.5	3 3 1.5	3 3 2.6	4 4 1.3	3 2 1.5
TRICHLORO-ETHENE	mean median range	4 4 2.5	3 3 2.5	3 2 1.8	9 5 0.2-6	3 2 2.19	5 1 0.1-10	5 1 0.5-19	6 4 2.12	4 1 1.9
TOLUENE	mean median range	24 24 18.27	22 22 10.36	21 23 11.27	27 20 7.65	32 25 19.68	120 113 22.292	35 22 15.98	23 22 20.25	25 22 16.46
UNDECANE	mean median range	5 4 3.8	5 6 3.7	4 4 2.4	4 3 1.8	6 7 2.8	5 4 2.10	4 3 2.12	7 7 2.7	4 4 5.12
2-VINYL-PYRIDINE	mean median range	1 0.5 0.4-3	1 0.3 0.1-4	3 2 0.2-9	0.9 0.3 0.1-3.5	6 2 0.1-23	8 0.6 0.4-27	2 2 0.4-5	1 0.9 0.3-2	3 1.4 0.6-7
O-XYLENE	mean median range	7 6 4.11	8 6 4.15	6 6 3.9	11 11 5.22	24 21 8.50	33 25 10.68	12 12 4.24	9 6 5.19	11 7 5.27
M,P-XYLENE	mean median range	35 26 18.63	38 24 14.83	28 17 15.55	60 66 23.94	111 109 34.228	191 149 49.328	60 60 14.138	45 33 17.92	69 37 25.170
Pooled VOC's*	mean median	139 118	148 126	117 92	164 156	291 271	822 898	206 168	141 119	214 154
										201 181

\*Pooled VOC's is the sum of the concentrations of all volatiles specifically identified in this study. It is not a measure of total volatiles.

2023380300

In order to assess the worst and best air quality for both smoking and non-smoking situations, Table 3 presents individual values for the minimum and maximum RSP observed for smokers' offices and for non-smokers' offices. So, for example, the 'minimum smokers' offices' data in the first column presents all of the analyte values for the single visit that gave the minimum RSP value ( $40 \mu\text{g m}^{-3}$ ). For this, data from Site 5, which is an open-plan space with one smoker and twenty-nine non-smokers, has been excluded from the considerations in order not to bias the comparisons.

Table 4 presents the same evaluation, except that it is based on the single visit corresponding to the minimum and maximum CO value observed.

The data is best evaluated by considering each analyte, or group of analytes, in turn.

#### Nicotine

In smokers' offices the median airborne nicotine level was  $3.4 \mu\text{g m}^{-3}$  (range  $0.6$  to  $26 \mu\text{g m}^{-3}$ ). From Table 2 it can be seen that the highest median for an individual site was  $18.1 \mu\text{g m}^{-3}$  for Site 2, whilst the lowest median was  $1.0 \mu\text{g m}^{-3}$  for Site 5. The nicotine data is of a similar order, though slightly lower in magnitude, as compared to other studies of offices (19, 21, 22). Numbers of cigarettes smoked during each visit were not identified in this study, because we did not wish to influence the occupants behaviour by either observing or questioning. The data show little correlation between nicotine levels and numbers of smokers present, or between nicotine levels and smoker density (the number of smokers present divided by the size of the room).

Some nicotine was found in the air of non-smokers' offices, though this was at a low level with a median value of  $0.6 \mu\text{g m}^{-3}$  (range  $0.1$  to  $2.1 \mu\text{g m}^{-3}$ ). Site 4 gave a median nicotine value of  $0.1 \mu\text{g m}^{-3}$ , whilst Site 10 gave a value of  $1.4 \mu\text{g m}^{-3}$ . Site 10 had been occupied by a smoker up to one week prior to the start of this investigation and hence it is possible that this level is due to a re-emission of nicotine from the furnishings. It is also possible that some nicotine is transferred through the air-conditioning system, or that unknown to us, a smoker occasionally visited this site.

#### RSP and UV-RSP

The RSP median value in smokers' offices was  $91 \mu\text{g m}^{-3}$  (range  $19$  to  $225 \mu\text{g m}^{-3}$ ). The UV-RSP median value, which is an estimate of the possible ETS contribution to RSP, was  $24 \mu\text{g m}^{-3}$  (range  $1$  to  $75 \mu\text{g m}^{-3}$ ).

Median values of RSP for each smoking site correlated poorly (0.522) with corresponding nicotine values. However, median UV-RSP values, again for each smoking site, gave an excellent correlation (0.962) with corresponding nicotine values. This indicates that the sum of the other particulate sources in this building is far more significant than the contribution from ETS (which may be, in smokers' offices, of the order of 30% of the total).

Data from non-smokers' offices yields a median RSP value of  $71 \mu\text{g m}^{-3}$  (range  $27$  to  $208 \mu\text{g m}^{-3}$ ). Some UV-RSP was also observed, with a median of  $8.8 \mu\text{g m}^{-3}$  (range  $1$  to  $17 \mu\text{g m}^{-3}$ ). The non-smoking Site 10 had a higher median RSP than smoking Sites 1, 3, 4, 6, 7 and 8, though the median UV-RSP for Site 10 was only higher than smoking sites 3 and 5.

Interestingly, Site 5 (the open plan area with 29 non-smokers and one smoker) had a higher than average median RSP value ( $129 \mu\text{g m}^{-3}$ ) but a lower than average UV-RSP value ( $7 \mu\text{g m}^{-3}$ ), indicating a significant non-ETS particulate source in this area.

The comparison of minimum and maximum RSP visits for smokers' and non-smokers' offices (Table 3) is interesting. Overall, there is a tendency for analyte levels to increase corresponding to the increase in RSP value. The increase is not of the same order of magnitude (apart from nicotine and UV-RSP in smokers' offices). Comparing benzene levels, for example, there is an increase from  $4$  to  $8 \mu\text{g m}^{-3}$  in smokers' offices, and an increase from  $15$  to  $18 \mu\text{g m}^{-3}$  in non-smokers' offices.

2023380301

TABLE 3: Data from a single visit corresponding to the minimum and the maximum RSP value obtained\*, separate by smokers' and non-smokers' offices.  
 (Data in  $\mu\text{g m}^{-3}$ , apart from CO and CO<sub>2</sub> which is given in ppm)

	Smokers' Offices		Non-Smokers' Offices	
	Minimum (Site 3)	Maximum (Site 2)	Minimum (Site 9)	Maximum (Site 4)
Nicotine	1.5	11	0.3	0.1
Respirable suspended particulates (RSP)	40	225	27	208
UV-RSP	3	72	3	4
Carbon monoxide	0.8	1.0	2.2	1.0
Carbon dioxide	550	500	500	600
Benzene	4	8	15	18
Chlorobenzene	0.1	0.3	0.4	0.3
n-Decane	24	4	8	5
1,2-Dichlorobenzene	0.2	0.5	0.4	0.7
1,2-Dichloroethane	4	10	20	18
Dodecane	2	2	1	2
Ethyl benzene	3	8	3	5
Limonene	3	4	4	4
n-Octane	3	2	4	2
$\alpha$ -Pinene	1	7	3	8
Styrene	4	15	10	12
Tetrachloroethene	3	6	4	8
Trichloroethene	2	5	1	0.2
Toluene	11	36	23	25
Undecane	8	3	6	4
2-Vinyl Pyridine	0.5	0.3	1.3	0.3
$\sigma$ -Xylene	6	15	7	11
m/p-Xylene	16	83	33	81

\* Site 5 has been excluded from consideration

2023380302

TABLE 4: Data from a single visit corresponding to the minimum and maximum CO value obtained, separated by smokers' and non-smokers' offices.  
 (Data in  $\mu\text{g m}^{-3}$  apart from CO and  $\text{CO}_2$  which is given in ppm)

	Smokers' Offices	Non-Smokers' Offices		
	Minimum (Site 3)	Maximum (Site 6)	Minimum (Site 9)	Maximum (Site 4)
Nicotine	2.0	4.2	0.8	0.1
Respirable suspended particulates (RSP)	50	33	43	71
UV-RSP	6	21	6	9
Carbon monoxide	0.5	3.4	0.7	3.6
Carbon dioxide	600	700	500	800
Benzene	6	48	12	23
Chlorobenzene	0.2	0.5	0.6	0.4
n-Decane	2	20	7	5
1,2-Dichlorobenzene	0.2	0.1	0.9	1.1
1,2-Dichloroethane	4	39	19	12
Dodecane	1	3	3	1
Ethyl benzene	2	54	4	13
Limonene	2	4	5	2
n-Octane	2	485	5	2
$\alpha$ -Pinene	1	6	6	7
Styrene	3	15	19	16
Tetrachloroethylene	1	5	4	4
Trichloroethylene	0.5	10	4	1
Toluene	22	292	22	65
Undecane	2	10	7	8
2-Vinyl Pyridine	9	0.6	3	4
$\sigma$ -Xylene	4	41	11	22
m/p-Xylene	17	149	68	94

2023380303

Repace and Lowrey (23) have suggested that a typical non-smoker working in an office building in the U.S. (generally air-conditioned), would be exposed to average concentrations of particulates due specifically to ETS of  $242 \mu\text{g m}^{-3}$  (range 100 to  $1000 \mu\text{g m}^{-3}$ ). On this basis, these authors proposed numbers of deaths in non-smokers due to exposure to ETS in the workplace and suggested that workplace smoking restrictions should be introduced. Our data, acquired in a relatively well ventilated but not untypical U.K. office building, suggests average ETS particulate levels some 1.0 times lower than those given by Repace and Lowrey; UV-RSP values in our study for smokers' offices being  $24 \mu\text{g m}^{-3}$  (range 1 to  $75 \mu\text{g m}^{-3}$ ).

It is known that Repace and Lowrey did not take into proper account particulate sources other than ETS, but rather suggested that ETS was the major source of particulates. Our study suggests that this might not be so. The work of other researchers in the U.S. also suggests that the Repace and Lowrey data may be a gross over-estimate (24).

#### Carbon Monoxide

Median carbon monoxide levels were 1.1 ppm (range 0.5 to 3.4 ppm) for smokers' offices, and 1.0 ppm (range 0.7 to 3.6 ppm) for non-smokers' offices.

Comparing median values for each office, Site 5 has the highest at 1.6 ppm. This is associated with a relatively high RSP value and a relatively low UV-RSP value for this Site, indicating a source other than ETS being responsible. All other sites were found to have similar median CO levels (range 0.9 to 1.4 ppm).

For smokers' offices alone, and excluding Site 5, there was a relatively good correlation between median CO level for each site and both the corresponding nicotine level (corr. 0.929) and the corresponding UV-RSP level (corr. 0.924). This correlation was not so well defined for corresponding RSP values (corr. 0.752). This suggests that ETS is contributing to the CO level in smokers' offices, though this contribution seems to be of the order of 0.1 to 0.4 ppm.

Table 4 compares four individual visits corresponding to minimum and maximum CO values for smokers' and non-smokers' offices. For smokers' offices the increase in CO from minimum to maximum corresponds to increases in many of the other analytes. However, Site 6 (where the maximum CO level was observed) was a drawing office where many print materials were being used, and so this confounds the interpretation. For non-smokers' offices, the CO level increase from minimum to maximum corresponded to an increase in particulate,  $\text{CO}_2$  and some (but not all) aromatic VOC levels, but a decrease in hydrocarbons and in some chlorinated VOCs.

The relatively low CO levels observed may, in part, be due to the fact that air is drawn into the building from roof level, well away from the traffic circulating the building.

#### Carbon Dioxide

Median  $\text{CO}_2$  levels for all smokers' sites was 600 ppm (range 450 to 1000 ppm) and 500 ppm (range 450 to 800 ppm) for non-smokers' offices. These levels suggest that the building is relatively well ventilated.

Comparing medians for each site office, Site 1 (a small office occupied by a single smoker) consistently had higher levels of  $\text{CO}_2$  than other sites. A comparison of Site 1 with Site 9 (a similarly small office occupied by a single non-smoker) suggest that the high  $\text{CO}_2$  levels in Site 1 are not a simple correlation with size of room. It was noticed, however, that in Site 1, ventilation inlets at the window sills were significantly obstructed and this may be of importance.

2023380304

### Volatile Organic Compounds

Some 18 VOC's were quantified in this study. One of the most interesting is benzene. The EPA's TEAM study (14) has suggested that ETS is a major source of benzene in indoor air. Our data from this office building does not confirm that suggestion. The median benzene level in smokers' offices was  $8 \mu\text{g m}^{-3}$  (range 3 to  $49 \mu\text{g m}^{-3}$ ), whilst the median benzene level in non-smokers' offices was  $10 \mu\text{g m}^{-3}$  (range 3 to  $31 \mu\text{g m}^{-3}$ ). The difference in benzene levels between smokers' and non-smokers' offices was not statistically significantly different at the 95% confidence limit.

It is found that there is poor correlation over individual smokers' offices (excluding Site 5) between median benzene levels and median nicotine (corr. 0.324) or between median benzene levels and median UV-RSP (corr. 0.243). Looking across all offices, Site 5 has the highest median benzene level ( $13 \mu\text{g m}^{-3}$ ) with Site 9 the second highest ( $12 \mu\text{g m}^{-3}$ ).

The absolute levels of benzene found in this study are similar to that found in the TEAM study, but the conclusions on what is the major source of benzene are quite different.

Comparing all of the median VOC values in Table 2, 9 VOC's are higher in non-smokers' offices, 7 are higher in smokers' offices and 2 are similar in both situations. However, levels are very similar for all VOC's comparing smoking and non-smoking offices. There is no indication that the presence of ETS is associated with significantly increased levels of VOC's in this office environment.

Comparing median values across individual sites, two offices stand out as being unusual. Site 5, which has relatively high RSP and CO values and low nicotine and UV-RSP values, has higher than average levels of benzene, styrene, o-and m/p-xylene, and chlorobenzene. No obvious source for these levels was identified (tobacco smoking was clearly not a major source). It is possible that this open plan area was not ventilated as efficiently as the smaller offices, though CO<sub>2</sub> levels do not confirm this.

Site 6 is also unusual. Much higher than average levels of n-decane, n-octane, ethylbenzene, toluene and xylenes were identified. The source of these chemicals is clear. This site is a drawing office working with various inks and printing material, and located adjacent to rooms processing photographic materials. Hence when pooled VOC's are calculated by adding the median concentrations of all the VOC's (including nicotine) together, Site 6 is seen to contain around 9 times more VOC's than many of the other offices.

It should also be noted that the peak toluene value averaged over one of the hour long visits to Site 6 was a quarter of the odour detection limit, and the peak styrene level of  $79 \mu\text{g m}^{-3}$  was equivalent to the odour detection limit. This styrene level was observed on just one occasion in Site 9 (non-smoking, single occupant) and was also associated with higher than averaged levels of benzene, chlorobenzene, ethylbenzene, toluene and xylenes.

In order to illustrate the number of volatile chemicals present in the air of smokers' and non-smokers' offices, Figure 2 compares chromatographic profiles taken in Site 3 and Site 4. The two chromatograms may be directly compared as the 100% ion count has been adjusted for the volume of air sampled in each visit. It is clear that the chromatograms are similar, apart from the peak corresponding to nicotine (representing  $5 \mu\text{g m}^{-3}$ ) being present in the sample from the smokers' office.

Figure 2

CHROMATOGRAPHIC PROFILES OF  
MULTIPLE OCCUPANT OFFICES

TOTAL ION COUNT

100%

ONE SMOKER IN  
OFFICE

nicotine

TOTAL ION COUNT

100%

NO SMOKERS IN  
OFFICE

nicotine

TIME (MIN)

1015

2023380306

### Cigarette Equivalent Calculations

Some authors (17, 19), have attempted to put the levels of ETS constituents into perspective through cigarette equivalent calculations. This exercise takes the median value of a constituent such as nicotine or UV-RSP and assumes that this is the constant exposure. A typical breathing rate and a time of exposure (e.g. the time spent in the office each day) is then used to arrive at a daily exposure to the constituent. This is then compared to the delivery of the relevant constituent (nicotine or particulate matter) that would be obtained from smoking one cigarette. Such calculations are strictly an estimate of exposure and not dose, are only relevant to the quantified constituent and not total ETS, and take no account of the differences between breathing air and inhaling smoke. However, with these facts noted, the calculations are still of interest.

So, if we assume a typical breathing rate at light work for a male adult of  $1.08 \text{ m}^3 \text{ h}^{-1}$  and for a female adult of  $0.62 \text{ m}^3 \text{ h}^{-1}$  (24, 25), an exposure time of 7 hours per day, 5 days per week, and typical mainstream deliveries from U.K. style cigarette of 1.3 mg per cigarette nicotine and 13.6 mg per cigarette particulates, then cigarette equivalent calculations can be made.

For nicotine, taking the median airborne nicotine value for smokers' offices as  $3.1 \mu\text{g m}^{-3}$ , then a non-smoker present all day in the office would, on this average, be exposed to the equivalent of 0.018 of a cigarette (male) or 0.010 of a cigarette (female). This means that a male non-smoker would have to work in the smoker's office for over 11 weeks before being exposed to the equivalent nicotine as from smoking one cigarette. For females, this time would have to be 20 weeks. In other words, a female non-smoker would have to work for over seven and one half years in the smoker's office before being exposed to the equivalent nicotine of smoking a pack of 20 cigarettes.

This is based on the median value. Even for the office with the highest median airborne nicotine (Site 2,  $18.1 \mu\text{g m}^{-3}$  nicotine), the nicotine cigarette equivalent values are 0.105 cigarette per day (male) or 0.06 cigarette per day (female).

If the calculation is based on UV-RSP as being an estimate of the ETS contribution to respirable particulates and taking the median value from smokers' offices as  $24 \mu\text{g m}^{-3}$ , then the non-smoker working all day in the smoker's office would be exposed to the equivalent particulates of 0.013 (male) or 0.0077 (female) cigarettes per day. This again would result in a male non-smoker working in the smokers' office for 15 weeks before being exposed to the equivalent particulates as smoking one cigarette. For females this equates to almost 26 weeks.

The highest median UV-RSP (Site 2,  $69 \mu\text{g m}^{-3}$ ) results in particulate cigarette equivalents of 0.038 cigarettes per day (male) or 0.022 cigarettes per day (female).

These calculations simply serve to illustrate that levels of ETS constituents in the smokers' offices of this relatively well ventilated building are extremely small.

### CONCLUSIONS

This investigation of chemicals in the air of smokers' and non-smokers' offices in an air-conditioned building results in several conclusions.

1. The levels of constituents related to ETS (nicotine and UV-RSP) in smokers' offices were found to be low, both in terms of industrial time weighted exposure limits and in comparison to other chemicals present in the air.
2. In cigarette equivalent terms, a non-smoking male would have to work in a smoker's office for an average 11 weeks before being exposed to the nicotine equivalent of one cigarette. Based on particulates, this time extends to 15 weeks. For females, this equates to 20 weeks (based on nicotine) or 26 weeks (based on particulates).

2023380307

3. Both medians and ranges of ETS-related particulate levels are some ten times lower than those suggested by Repace and Lowrey (23) to be typical of U.S. office buildings.
4. By comparing smokers' and non-smokers' offices, and by observing values obtained for RSP and UV-RSP, it seems that, in this environment, ETS was a minor contributor to respirable particulate levels in air.
5. The presence of ETS resulted in a slight increase in CO levels, of the order of 0.1 ppm.
6. ETS did not significantly contribute to levels of volatile organic compounds in office air. In direct conflict with the findings of the U.S. EPA TEAM study (14), our research suggests that tobacco smoking does not result in significant increases of compounds such as benzene in office air.

#### REFERENCES

1. C.W. Nystrom and C.R. Green, Proceedings of the ASHRAE Conference IAQ86, Managing Indoor Air for Health and Energy Conservation, Atlanta, Georgia, April 20-23, 1986, 213-233.
2. Committee on Passive Smoking, Board of Environmental Studies and Toxicology, National Research Council, Environmental Tobacco Smoke, Measuring Exposures and Assessing Health Risks, National Academy Press, Washington DC, 1986.
3. Report of the U.S. Surgeon General, Health Consequences of Involuntary Smoking, U.S. Government Printing Office, Washington D.C., 1986.
4. The Fourth Report of the Independent Scientific Committee on Smoking and Health, Her Majesty's Stationery Office, London, 1988.
5. American Cancer Society, General Facts on Smoking and Health, 3, 1988
6. K. Überla, Int. Arch. Occup. Environ. Health, 9, 21-37, 1987.
7. P.N. Lee, Misclassification of Smoking Habits and Passive Smoking. A Review of the Evidence, Springer-Verlag, Berlin, 1988.
8. T.D. Sterling and B. Mueller, Am. Ind. Hyg. Assoc. J., 49, 423-426, 1988.
9. K. Sexton and J.J. Wesolowski, Environ. Sci. Technol., 19, 305-309, 1985.
10. National Research Council, Board of Toxicology and Environmental Health Hazards, Indoor Pollutants, National Academy Press, Washington D.C., 1981.
11. A.S. Robertson, P.S. Burge, A. Hedge, J. Sims, F.S. Gill, M. Finnegan, C.A.C. Pickering and G. Dalton, B.M.J., 291, 373-376, 1985.
12. E. Noma, B. Berglund, U. Berglund, I. Johansson, and J.C. Baird, Atmos. Environ., 22, 451-460, 1988.
13. M.J. Finnegan, C.A.C. Pickering and P.S. Burge, B.M.J., 289, 1573-1575, 1984.
14. L.A. Wallace, E.D. Pellizzari, T.D. Hartwell, C. Sparacino, R. Whitmore, L. Sheldon, H. Zelon and R. Pernitt, Environ. Res., 43, 290-307, 1987.
15. C.W. Bayer and M.S. Black, J. Chrom. Sci., 25, 60-64, 1987.
16. M.W. Ogden, L.W. Eudy, D.L. Heavner, F.W. Conrad, and C.R. Green, presented at 40th Tobacco Chemists' Research Conference, Knoxville, TN, Oct 1986.

2023380308

17. G.B. Oldaker III, and F.C. Conrad, Environ. Sci. Technol., **21**, 994-999, 1987.
18. M.W. Ogden, 101st Annual International Meeting of The Association of Official Analytical Chemists, Sept 14-17, San Francisco, CA, Poster H240, 1987.
19. J.R. Carson and C.A. Erikson, Environ. Tech. Lett., **9**, 501-508, 1988.
20. C.J. Proctor, Environ. Tech. Lett., **9**, 553-562, 1988.
21. T.D. Sterling, H. Dimich and D. Kobayashi, J. Air Pollut. Control Assoc., **32**, 250-259, 1982.
22. S.K. Hammond, B.P. Leaderer, A.C. Roche and M. Schenker, Atmos. Environ., **21**, 457-462, 1987.
23. J.L. Repace and A.H. Lowrey, Environ. Int., **11**, 3-22, 1985.
24. A. Arundel, T. Sterling and J. Weinkam, Indoor and Ambient Air Quality, ed. R. Perry and P.W. Kirk, Selper Press, 1988, 242-251.
25. P.L. Altman and D.S. Ditmer, Respiration and Circulation, Federation of American Society for Experimental Biology, Bethesda, MD, 1971.

2023380309

removed by diffusion and other mechanical collection mechanisms, are effectively captured by electrostatic forces.

### References

- Harrison AW (1978) Quiescent boundary layer thickness in aerosol enclosures under convective stirring conditions. *J Colloid Interface Sci* 69:563-570  
 Yoshida T, Kousaka Y, Okuyama K (1979) *Aerosol science for engineers*. Power Co Ltd, pp 131-132

### Results from Surveys of Environmental Tobacco Smoke in Offices and Restaurants

G. B. Oldaker III, P. F. Perfetti, F. C. Conrad Jr., J. M. Conner, and R. L. McBride

### Summary

Surveys were conducted in several major cities in order to estimate the exposures of occupants in offices and restaurants to environmental tobacco smoke (ETS). Concentrations of ETS were estimated by measuring vapor phase nicotine and ultraviolet particulate matter, an empirically derived parameter providing an upper limit for the contribution of ETS to respirable suspended particles (RSP). Area samples were collected with portable air sampling systems (PASS), which are battery-powered devices contained in otherwise ordinary briefcases, a design allowing sampling to be performed unobtrusively. Nicotine was determined with gas chromatography and nitrogen specific detection. UV-PM was determined spectrophotometrically by analyzing methanolic extracts of RSP collected after separation at 3.5  $\mu\text{m}$  with an inertial impactor. For offices, mean concentrations of nicotine, UV-PM, and RSP were 4.8, 27, and 126  $\mu\text{g}/\text{m}^3$ , respectively. Mean concentrations of nicotine, UV-PM, and RSP for restaurants were 5.1, 36, and 126  $\mu\text{g}/\text{m}^3$ , respectively.

Within the past decade, environmental tobacco smoke (ETS) has emerged as a major issue within the general subject area of indoor air quality. The issue has acquired increased attention owing to recent reports by the U.S. Surgeon General [28] and the National Academy of Sciences [5]. Reviewing results from epidemiological investigations, these two bodies concluded that there exists a causal relation between exposure to ETS and incidence of lung cancer. This conclusion has elicited controversy for several reasons: results of the investigations vary in terms of epidemiological and statistical significance and overall quality; relative risks indicated are low within the context of epidemiological associations; consistent "dose"-response relationships are not observed (with dose inferred from questionnaires regarding spouses' smoking behavior); and the experimental designs of the investigations admit the strong possibility of influence by biases and confounding. A scientifically rigorous approach to clarify controversial issue calls for quantifying exposures and doses of the subject populations either included in or affected by epidemiological investigations [4]. Toward this goal, scientists with the tobacco industry have been engaged in developing and applying sampling technologies and analytical methodologies for assessing exposures to ETS in indoor environments. This paper summarizes progress in connection with surveying exposures in offices and restaurants, two important, public environmental categories. Results from one of the several surveys conducted have been presented earlier by Conner et al. [7].

2023380310

## Experimental Method

Surveys were performed in four major cities in the U.S. and Canada during the spring, summer, and autumn months. The cities all have populations greater than 100,000. At least 30 samples were acquired for each environmental category in each city. Offices were surveyed in all cities; restaurants were surveyed in three cities. Sampling was conducted either by scientists with the tobacco industry or by independent contractors.

### Selection of Sampling Sites

Offices were selected based upon the criterion that they be shared by two or more persons of whom at least one smoked. Office managers were informed of this criterion and given a description of guidelines regarding appropriate sampling locations. These guidelines were contained in a protocol prepared as part of the overall project effort. Based upon these, managers selected offices to be sampled. Sampling was performed during normal business hours. None of the offices had smoking restrictions.

Restaurants were selected from listings contained within telephone directories. Sampling was performed during normal lunch and dinner hours. None of the restaurants had smoking restrictions.

### Selection of Sampling Locations

Sampling locations within offices and restaurants were selected based upon a protocol's guidelines, which were derived from those described by Nagda and Rector [19]. The protocol was prepared in response to recommendations of the American Chemical Society [1] and, in addition, was patterned after quality Assurance Project Plans required by the U.S. Environmental Protection Agency for projects conducted by them [21].

### Sample Collection

Area samples were collected with Portable Air Sampling Systems (PASS) [16], which from the outside appear to be ordinary briefcases. During operation the PASS remains closed. An on-off switch is located beneath the briefcase's handle, and inlet and exhaust ports are fashioned of brass to match the briefcase's normal hardware. With these battery-powered devices, integrated samples are obtained for determining concentrations of vapor phase nicotine, respirable suspended particles (RSP), and ultraviolet particulate matter (UV-PM), an empirically defined measure providing an upper estimate of the contribution of ETS to RSP. Eudy et al. [9] and Eatough et al. [8] have reported that at least 90% of nicotine associated with ETS is in the vapor phase. The PASS is also equipped with three monitoring devices including a carbon monoxide monitoring system, a thermistor, and a pressure transducer, the latter two which enable volumetric results to be adjusted to actual conditions of temperature and pressure. Data provided by monitoring devices are stored in a data logger. (Efforts to reduce and interpret carbon monoxide data are in progress; consequently, carbon monoxide measurements are not discussed further here.)

The PASS's nicotine sampling system includes a sorbent tube containing XAD-4 resin connected with a short section of rubber tubing to a constant flow sampling pump

operated at 1 l/min. The major components of the sampling system for particulate matter species are an inertial impactor separating at 3.5  $\mu\text{m}$ , a filter assembly housing a Fluropore membrane filter, and a constant flow sampling pump operated at 2 l/min. The inertial impactor is sized to correspond to that employed in piezoelectric balances manufactured by TSI, Inc., St. Paul, MN.

Samples were collected for a minimum of 1 h in order to provide adequate material for the gravimetric determination of RSP.

### Analysis

Nicotine was analyzed with a method representing an enhancement of the method employed by the U.S. National Institute of Occupational Safety and Health (NIOSH) [20]. This enhanced method, which entails gas chromatography and nitrogen specific detection, has been described by Ogden et al. [22]. The gravimetric method for determining RSP was derived from the method described by Treitman et al. [27]. UV-PM was quantified according to the method described by Conner et al. [6]. For this method, filters employed for the RSP determination are extracted with methanol and the absorbance of the methanolic extract is measured spectrophotometrically at 325 nm. Masses of UV-PM are then interpreted with a standard calibration curve obtained from generating known concentrations of ETS in an environmental chamber [12]. The methods for determining RSP and UV-PM have been shown to be unbiased relative to piezoelectric balances [14].

To ensure further the quality of results, collaborative tests were conducted involving laboratories engaged in the surveys. Results from these tests have been reported by Ogden and Conner [23].

### Results and Discussion

Data from determinations of nicotine, UV-PM, and RSP for restaurants and offices associated with each city were analyzed statistically. These analyses indicated that each data set associated with each city was distributed log-normally. Moreover, these analyses

**Table 1.** Summary of results for measurements of ETS in offices and restaurants. Concentrations in  $\mu\text{g}/\text{m}^3$

	Nicotine	UV-PM	RSP
<b>Offices</b>			
Mean	4.8	27	126
Range	0-69.7 (n = 156)	0-287 (n = 125)	0-1,088 (n = 131)
<b>Restaurants</b>			
Mean	5.1	36	126
Range	0-23.8 (n = 170)	0-184 (n = 82)	0-685 (n = 83)

2023830311

showed that no statistically significant differences existed among the data sets for the cities. Consequently, results for each analyte were pooled and geometric means were computed. Results for offices and restaurants are summarized in Table I.

### Offices

For offices, nicotine results are consistent with those previously reported. Hammond et al. [11] found concentrations of nicotine in offices that ranged from 3.1 to 28.2  $\mu\text{g}/\text{m}^3$ . These researchers used personal sampling devices that collect nicotine on Teflon-coated glass fiber filters treated with sodium bisulfite. Muramatsu et al. [17, 18] employ personal sampling devices utilizing Uniport-S coated with silicon OV-17 for the collection of nicotine. They reported average nicotine concentrations ranging from 5.9 to 22.2  $\mu\text{g}/\text{m}^3$  for 8-h-samples collected in three offices. Weber and Fischer [29] reported much lower concentrations of nicotine in offices. These researchers, however, employed Cambridge filters to collect nicotine, and as Badre et al. [3] have observed, substantial losses of nicotine occur with this procedure.

The mean concentration of RSP, 126  $\mu\text{g}/\text{m}^3$ , as well as the range of concentrations, 0 to 1,088  $\mu\text{g}/\text{m}^3$ , are comparable to results reported by Weber and Fischer [29]. Using a piezoelectric balance, these researchers reported mean and maximum RSP concentrations of 170 and 1,130  $\mu\text{g}/\text{m}^3$ , respectively, for samples collected in 44 workrooms. This same measurement technique was used by Quant et al. [25] who reported average concentrations of RSP ranging from 36 to 89  $\mu\text{g}/\text{m}^3$  during daytime periods in three offices.

The UV-PM results strongly suggest that a substantial portion of the RSP measured originates from sources other than ETS. Thus, based upon comparisons of the tabulated means, UV-PM represents about 20% of the RSP. This observation points to RSP's lack of specificity and therefore its general inappropriateness for use as an indicator of ETS in settings outside of the laboratory.

### Restaurants

Results associated with restaurants are also consistent with results previously reported. Muramatsu et al. [17] collected eight 1-h-samples in five restaurants; they reported mean and maximum nicotine concentrations of 14.8 and 27.8  $\mu\text{g}/\text{m}^3$ , respectively. (Hinds and First [13] used Cambridge filters to collect nicotine and reported much lower concentrations of nicotine in restaurants. However, as was noted above, their results are presumed to be biased owing to low collection efficiencies of the filters.)

Survey results for RSP are comparable to results reported by Repace and Lowrey [26], who surveyed RSP with a piezoelectric balance in 10 restaurants. RSP concentrations ranged from 29 to 414  $\mu\text{g}/\text{m}^3$  for 13 sampling periods of times ranging from 2 to 40 min. These researchers attempted to assess the contribution of smoking to indoor concentrations of RSP and to demonstrate the validity of a model for estimating such contributions based upon number of occupants and room volume. A mean RSP concentration of 42  $\mu\text{g}/\text{m}^3$  was found in no-smoking sections and places where smoking was not seen to occur. In places where smoking occurred, a mean RSP concentration of 171  $\mu\text{g}/\text{m}^3$  was found. Although smoking was observed in all the restaurants surveyed by us, smoking was not continuous. Thus, mean results would be expected to fall between these two means, as indeed is the case.

The UV-PM results for restaurants are similar to those of offices. Thus, relative to the tabulated mean RSP, UV-PM makes about a 30% contribution.

### Estimation of Exposure to ETS

The results of these surveys show mean nicotine and UV-PM concentrations to be low. In order to place these results in a more convenient form for discussion and interpretation, many researchers have employed the cigarette equivalent concept [13, 17, 24, 26, 29]. Assumptions attending use of this concept have been described [28]. Here, exposures are estimated from the mean nicotine concentrations reported in Table I. (Exposures estimated from mean nicotine concentrations are higher than those computed from UV-PM results, with the assumption made that UV-PM is equivalent to "tar" as defined by the Federal Trade Commission.) Also assumed for estimation of exposures are a breathing rate of 8.61/min, which corresponds to miscellaneous office work [2], and a U.S. sales weighted average "equivalent cigarette" delivering 0.88 mg nicotine [10, 15]. Accordingly, estimated mean exposures for an eight-hour work day in an office is 0.02 cigarette equivalent and for a 1-h meal in a restaurant, 0.003 cigarette equivalent.

### References

1. ACS Committee on Environmental Improvement and Subcommittee on Environmental Analytical Chemistry (1980) Guidelines for data acquisition and data quality evaluation in environmental chemistry. *Anal Chem* 52:2242-2249
2. American Society of Heating, Refrigerating and Air Conditioning Engineers, Inc (1985) ASHRAE Handbook, 1985 Fundamentals, pp 8.11-8.12, Atlanta, GA
3. Badre R, Guillerm R, Abran N, Bourdin M, Dumas C (1978) Pollution atmosphérique par la fumée de tabac. [Atmospheric Pollution from Smoking]. *Ann Pharm Fr* 30:443-452
4. Committee on the Epidemiology of Air Pollution, Board on Toxicology and Environmental Health Hazards, Committee on Life Sciences, National Research Council (1985) Epidemiology and air pollution. National Academy Press, Washington, DC
5. Committee on Passive Smoking, Board on Environmental Studies and Toxicology, National Research Council (1986) Environmental tobacco smoke. Measuring exposures and assessing health effects. National Academy Press, Washington, DC
6. Conner JM, Murphy JM, Oldaker GB III, Green CR, Angel AL (1986) Development of a method for estimating the contribution of environmental tobacco smoke (ETS) to indoor respirable suspended particulates (RSP). Presented at 40th Tobacco Chemists' Research Conference, Knoxville, TN
7. Conner JM, Conrad FW Jr, McConnell BC, Ogden MW, Oldaker GB III, Perfetti PE, Stancill MW (1987) Results from surveys of environmental tobacco smoke (ETS) in offices and restaurants in New York City. Presented at 41st Tobacco Chemists' Research Conference, Greensboro, NC
8. Eatough DJ, Benner C, Mooney RL, et al (1986) Gas and particle phase nicotine in environmental tobacco smoke. Presented at 79th Annual Meeting of the Air Pollution Control Association, Minneapolis, MN
9. Eudy LW, Thome FA, Heavner DL, Green CR, Ingebrethsen BJ (1986) Studies of the vapor-particulate phase distribution of environmental nicotine by selective trapping and detection methods. Presented at the 79th Annual Meeting of the Air Pollution Control Association, Minneapolis, MN
10. Federal Trade Commission Report (1986) Tar, nicotine, and carbon monoxide of the 207 varieties of domestic cigarettes

202380312

11. Hammond SK, Leaderer BP, Roche AC, Schenker M (1987) Collection and analysis of nicotine as a marker for environmental tobacco smoke. *Atmos Environ* 21:457-462
12. Heavner DL, Thome FA, Eudy LW, Ingebretsen BJ, Green CR (1986) A test chamber and instrumentation for the analysis of selected environmental tobacco smoke (ETS) constituents. Presented at the 79th Annual Meeting of the Air Pollution Control Association, Minneapolis, MN
13. Hinds WC, First MW (1975) Concentrations of nicotine and tobacco smoke in public places. *N Engl J Med* 292:844-845
14. Ingebretsen BJ, Heavner DL, Angel AL, Conner JM, Steichen TJ, Green CR: A comparative study of environmental tobacco smoke particulate mass measurements in an environmental chamber. Accepted for publication in *J Air Pollut Control Assoc*, November 1987
15. Maxwell JC, Jr (1986) The Maxwell Report. Revised 1985 Year-End Sales Estimates for the Cigarette Industry. February 3, 1986
16. McConnell BC, Perfetti PF, Walsh RF, et al (1987) Development and evaluation of portable air sampling system (PASS) for environmental tobacco smoke (ETS). Presented at 41st Tobacco Chemists' Research Conference, Greensboro, NC
17. Muramatsu M, Umemura S, Okada T, Tomita H (1984) Estimation of personal exposure to tobacco smoke with a newly developed nicotine personal monitor. *Environ Res* 35:218-227
18. Muramatsu M, Umemura S, Fukui J, Arai T, Kira S (1986) Estimation of personal exposure to ambient nicotine in daily environment. Presented at International Experimental Toxicology Symposium on Passive Smoking, Esen, West Germany
19. Nagda NL, Rector HE (1983) Guidelines for monitoring indoor air quality. EPA-600/4-83-046
20. National Institute for Occupational Safety and Health Manual of Analytical Methods, Vol 3, Method No. S293
21. Office of Monitoring Systems and Quality Assurance, Office of Research and Development, U.S. Environmental Protection Agency (1980) Interim guidelines and specifications for preparation of quality assurance project plans. QAMS/005/80
22. Ogden MW, Eudy LW, Heavner DL, Conrad FW, Jr, Green CR (1986) Improved gas chromatographic quantitation of trace levels of environmental nicotine. Presented at 40th Tobacco Chemists' Research Conference, Knoxville, TN
23. Ogden MW, Conner JM (1987) Method of analysis for nicotine, respirable suspended particles (RSP), and ultraviolet particulate matter (UV-PM) in environmental tobacco smoke (ETS): Collaborative study. Presented at 101st Meeting of the Association of Official Analytical Chemists, San Francisco, CA
24. Oldaker GB III, Conrad FW, Jr (1987) Estimation of effect of environmental tobacco smoke on air quality within passenger cabins of commercial aircraft. *Environ Sci Technol* 21:994-999
25. Quant FR, Nelson PA, Sem GJ (1982) Experimental measurements of aerosol concentrations in offices. *Environ Internat* 8:223-227
26. Repace JL, Lowrey AH (1980) Indoor air pollution, tobacco smoke, and public health. *Science* 208:464-472
27. Treitman RD, Spengler JD, Tosteson T (1981) Exposure assessment project. Quality assurance project plan. Research Triangle Institute, U.S. Environmental Protection Agency, Research Triangle Park, NC
28. U.S. Department of Health and Human Services (1986) The health consequences of involuntary smoking, Rockville, MD
29. Weber A, Fischer T (1980) Passive smoking at work. *Int Arch Occup Environ Health* 47:209-221

20230833023

## Strategy for Future ETS Exposure Measurements Relative to Its Transient Nature and Other Indoor Air Pollutants

I. O'Neill

### Introduction

From the outset, it has not been possible to relate exposure to individual components of mainstream (MS) and environmental tobacco smoke (ETS)<sup>1</sup> with important biological outcome in humans. We do know from the extensive studies by Spengler et al. (1985), Wallace et al. (1985), Hirayama (1981), both that ETS is a main indoor source of airborne particulates and volatile carcinogens, and also that passive exposure to tobacco smoke seems associated with increased risk of lung cancer. Following on the IARC evaluation of ETS carcinogenicity<sup>2</sup> (IARC, 1986), there are substantial problems in devising the appropriate strategy for exploring the relationship between ETS pollution and the biological outcome in humans. Looking at this another way, there is a fundamental gap between the epidemiological and experimental approaches to this problem. Discussed below are the key problems of the multiplicity of pollutants in indoor air and the potential importance of active although transient components; this discussion is related to some IARC activities in this field.

### Biologically Active Components of Indoor Air Pollution

Since most people spend 75% to 90% of their time on average breathing indoor air (NRC 1981), and building design has changed greatly in recent years (Mage and Gammie 1985), a number of recent studies have examined the biological activity of indoor air and substances infiltrating from outside. Mutagenicity of indoor air has been compared to indoor activities in studies in the Netherlands (Van Houdt et al. 1984), USA (Lewtas 1982), Finland (Salomaa 1987) and Norway (Lofroth et al. 1983). It is notable, however, that the levels of airborne particulate found, for example, by Spengler et al. (1985) in the USA are much lower than those arising from unventilated combustion of biomass in non-industrialized countries (WHO 1984) where respiratory illness is often a major contributor to mortality for women who are almost entirely nonsmokers. Hence, there

<sup>1</sup> The Sixth World Conference on Smoking and Health (Tokyo, 9-12 November, 1987) resolved that ETS is a misnomer implying a natural component of the environment. In the absence of any proposed alternative, ETS, however, is used in this paper.

<sup>2</sup> This IARC working group evaluation included the following statement: "The observations on nonsmokers that have been made so far are compatible with either an increased risk from "passive" smoking or an absence of risk. Knowledge of the nature of sidestream and mainstream smoke, of the materials absorbed during "passive" smoking, and of the quantitative relationships between dose and effect that are commonly observed from exposure to carcinogens, however, leads to the conclusion that passive smoking gives rise to some risk of cancer".

## Harvard's Indoor Air Pollution Health Study

J. D. Spengler

### Introduction

It is well established that indoor sources of air pollutants in homes, offices, public buildings, and transportation vehicles can result in higher indoor concentrations of contaminants. However, in epidemiologic studies it may not be entirely sufficient to characterize subject exposure with a description of sources and source use. Emission rates vary, source use varies, and air exchange rates differ among homes, as well as other structures. Therefore, questionnaires will not be adequate for many contaminants. This point is illustrated by the distribution of annual indoor home concentrations of NO<sub>2</sub>, shown in Fig. 1.

One-hundred gas cooking homes in Portage, Wisconsin were monitored using integrating passive samplers. Annual concentrations range from 15 µg/m<sup>3</sup> to 150 µg/m<sup>3</sup>. While a question about cooking fuel differentiates mean concentrations in gas versus electric homes, descriptive questions cannot predict indoor concentrations within a gas cooking home.

Recognizing the limitation of questionnaires, we designed a more comprehensive indoor air pollution survey to characterize exposures in a multi-city air pollution health study. Several personal exposure studies have demonstrated that indoor (home) concentrations of respiratory particles, nitrogen dioxide, CO etc. are predictive of personal exposures [1-4]. Therefore, a microenvironmental monitoring approach was adapted for characterizing exposures to elementary school-age children. Homes, schools and ambient environments were monitored for respiratory particles (PM 2.5) and nitrogen dioxide (NO<sub>2</sub>).

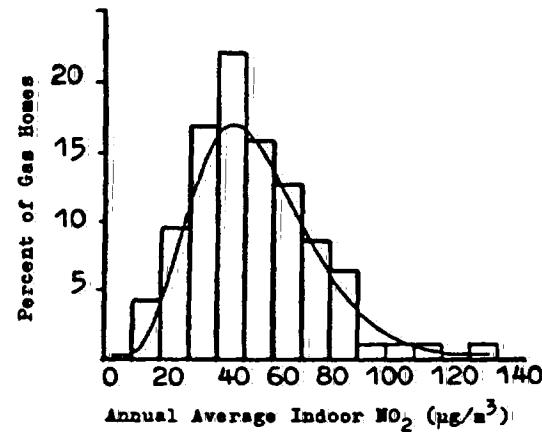


Fig. 1. Distribution of annual mean indoor NO<sub>2</sub> concentrations for approximately 100 gas cooking homes in Portage, WI

This paper describes the design and preliminary results of Harvard's Indoor Air Pollution Health Study. This study was conducted in six U.S. cities between 1984 and 1988: Watertown, MA; Kingston, TN; St. Louis, MO; Steubenville, OH; Portage, WI; Topeka, KS. Approximately 1,000 children in the 3rd to 5th grades in each community were administered two annual Health Characterization Questionnaires (HCQ) through the schools. In addition, pulmonary functions were measured. Using the first HCQ, homes were stratified on the presence of sources for NO<sub>2</sub> and particulate matter. The indoor environments of approximately 300 homes were monitored over a year. Children in these homes participated in a diary survey of daily respiratory symptoms.

### Design Overview

The first annual HCQ was administered through the school system to children in grades 3 to 5. Parents completed a questionnaire that includes a history of respiratory illnesses, respiratory symptoms over the previous 12 months, and several items on potential home factors. Pulmonary function measurements were made on the children in the schools. Details are presented elsewhere [5, 6]. A second HCQ and exam was administered approximately one year later.

Using the results of the first HCQ, homes were stratified based on the presence or absence of gas cooking and parental smoking. In most of the communities four cells were selected. The homes were randomly selected within these cells to participate in a year-long respiratory health diary study. The cells had unequal weights because concentrations within homes with sources had been shown to have greater variation. In Kingston, Tennessee and Portage, Wisconsin, additional stratification was based on wood and kerosene heating fuels.

Approximately 350 children were recruited to participate in a respiratory symptoms diary study starting in October. Mothers were instructed on how to use a calendar to record symptoms and severity from a set of predefined conditions. Table 1 lists symptoms that were recorded on the calendar. Approximately every two weeks the mother was called to read off symptoms. At the end of each month, calendars were returned to Harvard for entry and for comparison to the data obtained by telephone. Preliminary analysis of symptoms have identified three clusters of symptoms. One set of symptoms appears to reflect upper respiratory tract infection, another reflects lower respiratory tract infection, and a third allergic conditions. Table 1 lists symptoms and severity of symptoms included in the diary. Only preliminary health analysis has been performed because the study has not been completed for all cities.

The exposure component consisted of a core set of parameters that were measured in all homes. Measurements consisted of integrated week-long NO<sub>2</sub> and particle samples

Table 1. Symptoms included in Harvard's respiratory health diary

Hoarseness	Ear pain or discharge
Sore throat	Runny or stuffed nose
Cough	Burning, aching or red eyes
Phlegm from the chest	Restricted activities
Pain in the chest	Saw doctor or nurse
Wheezing	Hospitalized
Fever	Healthy or none of the above

2023380315

collected during the winter and summer. School and outdoor measurements were made to the complete assessment of the primary microenvironments for children.

#### Indoor Air Quality Assessment

A large variety of contaminants have the potential to cause or aggravate respiratory symptoms. However, the primary focus of this Harvard study was two combustion sources: tobacco and cooking fuel. Thus, particulates and nitrogen dioxide were measured in every home to derive an exposure estimate for each child.

It was recognized that other contaminants might be important contributors to respiratory symptoms or might be confounding results. Additional substudies were performed. First, questions about mold, mildew and moisture were included in the follow-up HCQ. Preliminary analysis indicated significant association among these factors and respiratory symptoms. Fungal and bacteria sampling were then performed in a subset of homes, in four cities, to examine the relationship among these categorical descriptions and actual contamination levels.

Substudies were performed to determine the impact of woodburning stoves and kerosene heaters. These sources were more prevalent in two of our communities: Kingston, TN and Portage, WI.

In order to quantify the impact of sources to indoor air pollution, additional investigations were conducted. A subset of homes (approximately 30) were monitored for two weeks in each of the four seasons. Air exchange measurements were made using a tracer gas method. Further, in several hundred homes the particulate samples were analyzed for elemental composition, ionic concentrations of sulfates and nitrates.

The monitoring protocol required two winter weeks (November–March) and two summer weeks (May–August) of monitoring in each home. Previous year-round studies had indicated that between 70 and 90 percent of the pollution concentration variance within a home type was accounted for by capturing the seasonal variations. Nevertheless, a set of 30 homes were monitored in each season. Particulate samplers had pre-separation impactors to collect only particles less than  $2.5 \mu\text{m}$  diameter [7]. The device had 14 day times that were set to operate when the child was expected to be at home. This was approximately 128 hours per week. Filters were changed each week. Thus, most homes had four week-long particle samples. Integrating, passive  $\text{NO}_2$  diffusion tubes were placed in the kitchen, living room, bedroom and outdoors [8]. For the first three communities the integration time was one week. Because the week-to-week correlation was very high ( $R^2 > 0.75$ ) the sampling times were changed for the last three communities to two weeks for both winter and summer.

Passive water vapor tubes were also placed in each home. These one-week integration tubes provide a measure of moisture content of the air in a home that can be converted to relative humidity.

In a subset of approximately 100 homes per city, air exchange rates were estimated from the dilution of a conservative tracer gas that was being leaked continuously into the home [9]. Sources of a non-reacting perfluorocarbon molecules were placed in a central region of the home, and up to four passive charcoal collections were placed in various rooms of the home. Whole house air exchange rate was calculated by the average across the interzonal concentrations.

2023380316

## Results

This research is ongoing and only partial results are available at this time. Summarized in this paper are the concentrations of NO<sub>2</sub> and respirable particles sampled in five of the six cities. This paper highlights the findings for several of the substudies conducted.

### Nitrogen Dioxide

The ambient sources of nitrogen dioxide are automobiles and fossil fuel combustion from power plants and industries. Auto exhaust is the primary source in our six communities. Ambient concentrations ranged from a city mean of 8 ppb in Portage, Wisconsin to 24 ppb in St. Louis, Missouri. There was a pattern of lower ambient concentrations in the smaller, more rural towns versus larger metropolitan areas. The percent standard deviation of the outdoor measurements were between 15 and 20 percent. The rural communities had greater spatial variation in NO<sub>2</sub> that reflected differences in auto traffic density. Similarly, higher concentrations were noted near intersections and heavily travelled roadways in the larger communities.

Indoor sources of NO<sub>2</sub> included gas cooking fuels, unvented kerosene and gas heaters, and faulty gas furnaces and water heaters. The influence of gas cooking on indoor concentrations is most notable. In homes with gas cooking, the kitchen concentrations are higher than the activity room and bedroom concentrations. The bedroom and activity

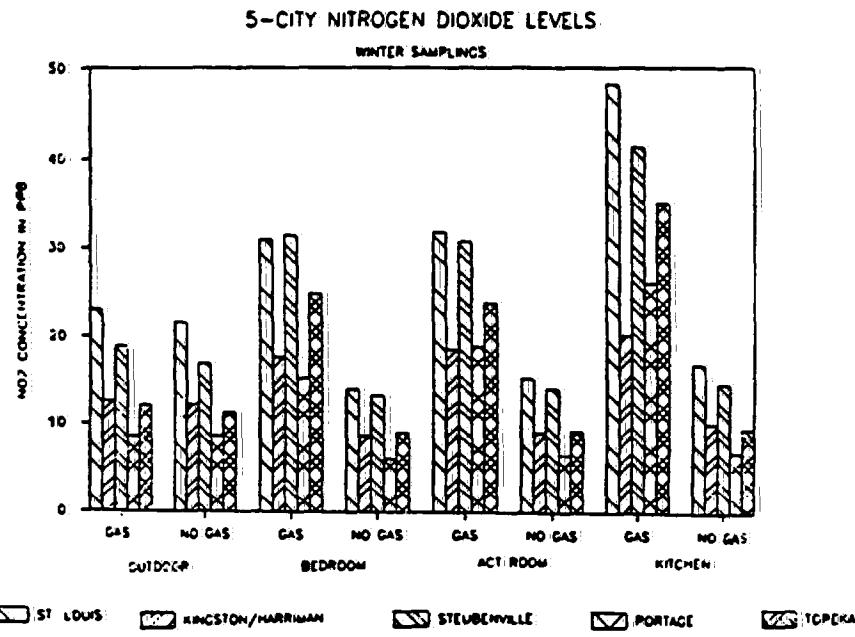


Fig. 2. Mean winter indoor NO<sub>2</sub> (ppb) concentrations inside and outside homes in five communities

2023380317

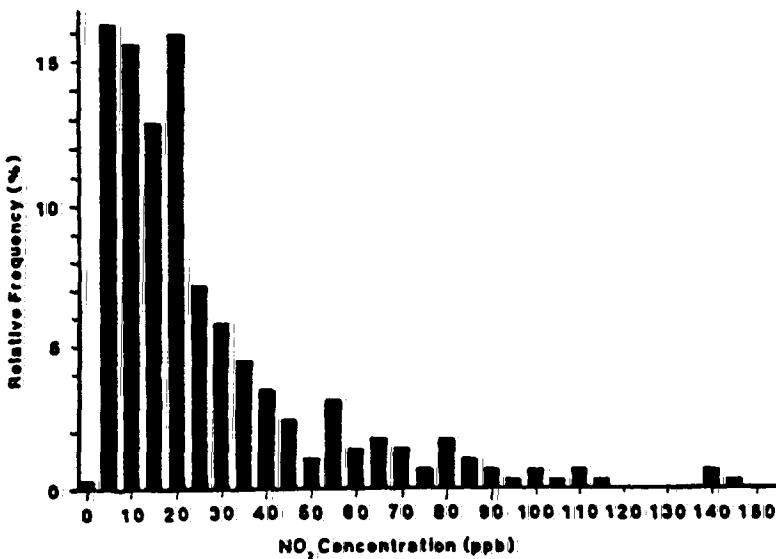


Fig. 3. Distribution of NO<sub>2</sub> levels (ppb) in Kerosene heater substudy homes

room have similar concentrations. In non-source homes the indoor concentrations are determined by outdoor concentrations. Levels are always lower because NO<sub>2</sub> will either react or be absorbed on indoor surfaces. For gas cooking homes, winter indoor concentrations are higher than summer concentrations. The opposite is true for non-source homes. This reflects the differences in the air exchange rate between winter and summer for most homes. Figure 2 presents the overall mean concentrations for NO<sub>2</sub> for the winter sampling period in five cities. Kingston, TN is not shown because there were very few gas cooking homes. This illustrates the differences among cities, differences among rooms within a home, and differences between home types.

#### Kerosene

Unvented gas and kerosene heaters are a major source of indoor NO<sub>2</sub>. Kingston/Harriman, Tennessee had more kerosene heaters than any of the other communities. They are banned in some states and less likely to be used in urban areas. With the assistance of the Oak Ridge National Laboratories a special study was conducted to assess the indoor NO<sub>2</sub> concentrations associated with kerosene heater use. Residents were given a set of samplers to measure NO<sub>2</sub> over ten consecutive weeks during the winter. In a few homes continuous monitoring equipment was used to document short-term concentrations. Figure 3 displays the frequency distribution of week-long NO<sub>2</sub> concentrations in homes with kerosene heaters. The range of concentrations reflects differences in heater type, actual usage, home and room volumes, and air exchange rate. Note that approximately 21% of the weeks had concentrations exceeding 50 ppb. The short-term concentrations are even higher. It is not uncommon for 1-h NO<sub>2</sub> concentrations to exceed 200 ppb during kerosene burner use. Figure 4 plots the combined NO and NO<sub>2</sub>

2023380318

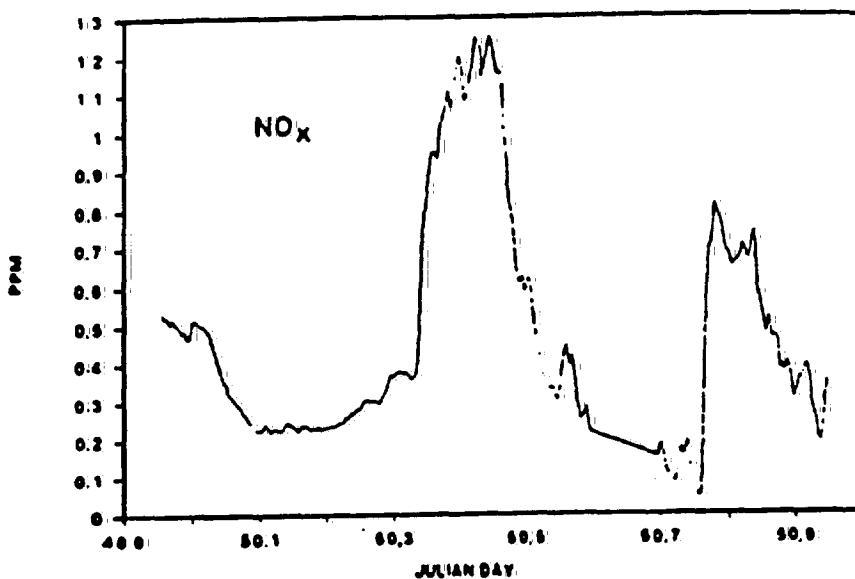


Fig. 4. One day of time-dependent pollutant levels from house 6476 during February

concentrations (ppm) inside one home that had a kerosene heater operating intermittently. Note that concentrations often exceed 500 ppb.

#### *Respirable Particles*

There are numerous sources of indoor and outdoor particulates. From previous studies we learned that outdoor fine particles penetrate into homes. The penetration ratio can be estimated by examining the indoor and outdoor concentration of elements and/or compounds that are outdoor contaminants that are introduced into the indoor air once they settle out or react with surfaces. Sulfate particles and elemental lead are good tracers for submicron size particles of outdoor origin. The penetration of outdoor particles varies by season and by home. During the winter the penetration factor is typically 0.4 and during the summer it is typically 0.8. Homes that are airconditioned or are constructed to have low air exchange rates have lower penetration values.

Indoor sources originate from a variety of activities including vacuuming, cooking, aerosol sprays and general human and pet activities. Quantifying the mass contributions of these activities is not possible in our large scale study that collects time integrated concentrations. There are other sources of indoor particle contamination. Tobacco smoke, kerosene heaters, woodburning stoves, cool mist and ultrasonic humidifiers can contribute to indoor particle concentrations. Tobacco smoke is the largest and most consistently identified source of indoor pollution in our study.

The indoor PM 2.5 particle concentrations in nonsmoking homes range between 19  $\mu\text{g}/\text{m}^3$  for Portage to 32  $\mu\text{g}/\text{m}^3$  for Kingston during the winter. In the summer, ambient PM 2.5 concentrations are higher because of secondary aerosol formation (sulfates). The indoor concentrations in non-airconditioned homes reflect this increase. However,

2023380319

Table 2. Particle concentrations indoors

		Winter			Summer		
		N	Mean	SD	N	Mean	SD
Watertown	Nonsmoking	78	21	9	72	19	7
	Smoking	176	53	24	168	33	18
St Louis	Nonsmoking	120	26	18	101	23	8
	Smoking	172	59	27	169	46	21
Steubenville	Nonsmoking	120	21	13	112	28	13
	Smoking	188	47	32	168	46	23
Portage	Nonsmoking	167	19	18	167	14	5
	Smoking	148	47	38	146	29	19
Kingston	Nonsmoking	162	32	28	164	25	14
	Smoking	137	73	44	123	51	30

overall there is only a slight effect. Indoor concentrations in Portage nonsmoking homes are still low,  $14 \mu\text{g}/\text{m}^3$ , but the indoor concentrations in Steubenville increased by  $7 \mu\text{g}/\text{m}^3$  to  $28 \mu\text{g}/\text{m}^3$  during the summer (Table 2).

Indoor particulate concentrations in homes with smokers are higher than for homes without smokers. The excess due to tobacco combustion varies by season and by city. Winter concentrations are higher than summer concentrations. During the winter, homes with smokers in St. Louis have  $22 \mu\text{g}/\text{m}^3$  more particulate mass than homes without smokers. In Kingston the excess is  $40 \mu\text{g}/\text{m}^3$ . Other cities are intermediate. During the summer, tobacco smoke contributes an additional mass concentration of  $16 \mu\text{g}/\text{m}^3$  in Portage and up to  $26 \mu\text{g}/\text{m}^3$  in Kingston. The summer difference may reflect the increase in air exchange.

Regression of indoor particulate concentrations on several home factors indicates that only the number of cigarettes smoked and inverse home volume are important. For an over-all impact, a cigarette smoked within a home contributes approximately  $1 \mu\text{g}/\text{m}^3$  to the integrated respirable particulate concentrations.

### Preliminary Health Results

Since the indoor air quality and diary symptom study has just been completed in September 1988, no results are available. However, the symptoms reported in the annual health questionnaire have been examined. The questionnaire included questions about smoking, heating and cooking fuels, heating appliances, and moisture and mildew conditions in the home.

Passive smoke exposure was prevalent in this sample of 6,273 children. Approximately 62% of the children were exposed to passive smoke in the home. The percentage ranged from 41.5% in Portage to 65.1% in St. Louis. The relative odds of respiratory symptoms were calculated for four categories of smoking, controlling for age, sex, parental education, gas cooking, wood stoves, and kerosene heaters. There was a general positive trend of increasing symptoms. When calculating the relative odds normalized to one

2023380320

pack/day smoking, all symptoms were significantly greater than one, except bronchitis and other non-respiratory illnesses. The increase in reported symptoms range between 15% for chronic cough to 24% for wheeze.

Wood stoves were associated with increased relative odds for respiratory illnesses, 1.32 (95% CI 0.99 to 1.76). Other symptoms were not significantly greater for children in homes with wood stoves. The only positive association of respiratory symptoms and gas cooking fuel was for doctor diagnosed respiratory illness before the age of two-relative odds 1.09 (95% CI 0.89 to 1.33). A similar estimate for the same symptoms is reported for homes with kerosene heaters, 1.13 (95% CI 0.88 to 1.44). However, kerosene heaters are only prevalent in one city, Kingston (33.1%).

The health questionnaire included questions concerning moisture in the home. The presence of molds, mildew and previous water damage were assessed. In five of the six cities at least one of these conditions was reported in more than 50% of the homes. There was a consistent and statistically strong association among respiratory symptoms and "moisture conditions." After adjusting for smoking, age, sex, city and parental education, the relative odds ratio varied from 1.27 to 2.12 for all respiratory symptoms including asthma and hay fever. After removing asthmatic children the relationships were even stronger.

### Preliminary Conclusions

Given the prevalence of smoking (approximately 60%) and "moisture" related problems (approximately 45%) in U.S. homes surveyed in this analysis, a large number of children may be at risk of increased respiratory illness. The associations between respiratory symptoms and parental smoking is similar across all cities and consistent with an exposure response function that increases with the number of cigarettes reported. Indoor particulate concentrates also increase in proportion to the number smoked at home per week.

The evidence associating molds, mildew and dampness with increased respiratory illness is consistent across six different communities. Unfortunately, the relationship between questionnaires and actual indoor concentrations of microorganisms (fungal spores) has not been fully characterized. Therefore, while strongly suggestive, we cannot say with certainty if there is an environmental agent responsible.

By comparison, the evidence for effects associated with other combustion sources indoors is not as strong. However, linking actual indoor concentrations ( $\text{NO}_2$ , particles) to the respiratory illness diaries of each child should increase statistical power.

### References

1. Spengler JD et al (1985) Environ Sci Technol 19:700-707
2. Quackenboss et al (1986) Environ Sci Technol 20:775-783
3. Spengler JD, Soczek (1984) Environ Sci Technol 18:268A-280
4. Akland et al (1985) Environ Sci Technol 19:911-918
5. Dockery et al (1985) Am Rev Respir Dis 131:511-520
6. Ware et al (1984) Am Rev Respir Dis 129:366-374
7. Marple et al (1987) Air Poll Cntrl Assoc 37:1303-1307
8. Palmes et al (1976) Am Ind Hyg Assoc J 37:570-577

2023380321

NOTICE  
This material may be  
protected by copyright  
law (Title 17 U.S. Code).

# INDOOR-OUTDOOR RELATIONSHIPS FOR PARTICULATE MATTER: EXPOSURE CLASSIFICATIONS AND HEALTH EFFECTS

James J. Quackenboss and Michael D. Lebowitz

Division of Respiratory Sciences, University of Arizona, Health Sciences Center, Tucson, AZ 85724,  
USA

Cliff D. Crutchfield

Dept. of Toxicology, University of Arizona, Tucson, AZ 85724, USA

EI 87-320 (Received 6 November 1987; Accepted 3 February 1989)

As part of a study on the effects of indoor and outdoor air pollutants on respiratory health, measurements of indoor inhalable ( $PM_{10}$ ) and respirable ( $PM_{2.5}$ ) particulate matter have been collected in a sample of exposure-classified households. There was a close relationship between average indoor PM in these two size ranges, with a slope of 1.08 ( $PM_{10}$  to  $PM_{2.5}$ ), intercept of 12.5  $\mu\text{g}/\text{m}^3$  and  $R^2$  of 88.6%. Samples collected in the same household during sequential weeks were generally closely related ( $R^2$  of 85% for both sizes; difference was nonsignificant), although week-specific activities were important in explaining difference within some homes. The median indoor/outdoor (I/O) ratio was 0.63 for homes without reported smoking, and 1.1 for those with smoking. Corresponding mean indoor-outdoor differences were -3.6 and +13.5  $\mu\text{g}/\text{m}^3$ , which was only significant for homes with smoking ( $p<0.01$ ). Indoor  $PM_{10}$  over 50  $\mu\text{g}/\text{m}^3$  was associated with non-specific (annoyance) symptoms.  $PM_{2.5}$  over 15  $\mu\text{g}/\text{m}^3$  was related to symptoms of acute respiratory infections (depending on age group) and to daily variability in peak flow rates (independent of age and sex). These effects may be related to environmental tobacco smoke (ETS) exposures that are correlated with the measured PM concentrations, although more specific indicators of ETS are needed to confirm this.

## INTRODUCTION

A major difficulty in determining the effects of particulate matter on human health has been the inability to adequately characterize exposures to air pollutants from both indoor and outdoor sources, and thereby to properly assign exposure classifications to individuals. The importance of assessing indoor pollutants as part of air-pollution health-effects studies has often been noted. Several studies have indicated that indoor exposures and indoor sources account for the largest proportion of an individual's exposure to several pollutants, including particulate matter (Dockery and Spengler 1981a; Spengler et al. 1985). These studies have been conducted in areas with climates cooler than that of the southwestern United States, and with correspondingly different housing stocks. Greater infiltration of outdoor

PM indoors and removal of indoor-generated PM may make outdoor PM levels more important, especially for small particles ( $<10 \mu\text{m}$ ;  $<2.5 \mu\text{m}$ ), than in other regions, where the infiltration of outdoor PM indoors is restricted (Dockery and Spengler 1981b). Previous studies conducted in Tucson, Arizona (Lebowitz et al. 1985) indicate a stronger relationship between total suspended PM outdoors and health effects relative to indoor sources than have studies conducted in other areas.

The type and size distribution of PM outdoors depends on its sources. Suspended outdoor PM derives from the combustion of fossil fuels and from natural sources. In the arid environment of Tucson, 80% of the PM is silica quartz, most of which averages around 5  $\mu\text{m}$  in mean aerodynamic diameter, and is suspended by vehicles operating over unpaved

2023380322

surfaces (Lebowitz 1984). Levels of total suspended PM exceed the NAAQS every year in several locations in the Tucson basin.

Indoor PM can be generated in significant amounts by tobacco smoking, wood burning, resuspension of dust and pollen through occupant activities, and other sources. Preliminary evidence indicates that indoor PM produces both vasomotor rhinitis and regular irritant symptoms, and that it interacts with gases to produce symptoms and to decrease pulmonary function. Other studies have shown specifically the effect on sensitive individuals of PM from both smoking and other sources in producing symptoms and decreasing peak flow (Lebowitz et al. 1985).

Since responses to pollutants of indoor and/or outdoor origin may be responsible for irreversible physiological changes and disability, the need to determine their relative contributions to total exposure is important for both regulatory and control purposes. The ability to collect and characterize particulate matter from several locations is important to help in this identification. Source-receptor techniques have been suggested for characterizing indoor PM, although further development of these techniques and identification of source-specific characteristics are required (Watson and Chow 1985).

## METHODS

The study population and design are described in a companion paper (Quackenboss et al. 1989). Briefly, a nested design was used to first characterize a larger

population using questionnaires, and then to make direct measurements of health and environmental factors on risk- and exposure-classified subgroups. Homes in the exposed and control groups were matched and sampled during concurrent weeks within a geographic cluster. Based on previous monitoring and source inventories, the geographic clusters were developed to provide relatively homogeneous average levels of PM and pollen. This paper describes the methods used to classify households by sources of particulate matter and the subsequent sampling in households used to evaluate these exposure classifications and indoor-outdoor relationships for PM.

Three PM classification groups, based on the estimated contribution of tobacco smoke (pipes and cigars were assumed to yield the same PM levels as four cigarettes) and wood burning to indoor respirable particle ( $PM_{2.5}$ ) concentrations, were derived from the basic questionnaires. The composition of these groups is shown in Table 1. Tobacco smoking was reported in 40% of the households, with the following breakdowns by cigarette-equivalents per day: 17.6% with less than 10/day (low), 13.3% with 11-20/day (medium), 9.1% of the households with more than one pack per day (high).

The total estimated contribution of wood smoke to indoor PM is also shown in Table 1. More than 30% of the households reported having and regularly using fireplaces: 11.4% reported using them only once per week in the heating season (low), 9.2% reported using them two or three days per week (medium), and an additional 9.2% reported using them more fre-

Table 1. Composition of PM classification groups based on estimated contribution of tobacco and wood smoke to indoor PM.

Estimated PM from Wood Smoke		Estimated PM ( $\text{mg}/\text{m}^3$ ) from Tobacco Smoke				Total
		None (20)	Low (20-40)	Medium (40-60)	High (60)	
None (20)	n	922	280	210	151	1563
	%	39.8	12.1	9.1	6.5	67.5
Low (>20-25)	n	177	42	30	16	265
	%	7.6	1.8	1.3	0.7	11.4
Med. (>25-40)	n	144	40	36	19	239
	%	6.2	1.7	1.6	0.8	10.3
High (>40)	n	147	46	32	25	250
	%	6.3	2.0	1.4	1.1	10.8
Total	n	1390	408	308	211	2317
	%	60.0	17.6	13.3	9.1	100.0
Missing	n	5				

2023300525

quently or often noticed smoke entering the room while using them (high). The use of wood stoves was reported in fewer households, with only 3.7% using them more often than once a week during the heating season.

#### PM samples

PM Samples were collected using the Harvard-designed Indoor Air sampler (Marple et al. 1987; Turner and Spengler 1985). This unit consists of a pumping and control unit (mass flow controller, timer, and elapsed time meter), time share unit (timer and controller to permit sequential collection by two samplers using the same pump), and the size-selective aerosol impactors for inhalable ( $<10 \mu\text{m}$ ) and respirable ( $<2.5 \mu\text{m}$ ) particle collection. The pumping unit operates at a low flow rate (4 L/min.); particulates are collected onto teflon filters for mass determinations and subsequent analysis (e.g., XRF). Flow rates were checked before and after sampling by using a calibrated rotameter and by checking the mass flow controller voltage. Particulate filters were pre- and post-weighed following standardized operating procedures for balance calibration, filter conditioning, and filter weighing. Quality control checks included limit checks for flow rates and filter weights and range checks of concentrations in relation to sample type and location. Outdoor, ambient air-quality data in each geographic cluster corresponding to the sampling days in each home were obtained from Pima County's Air Quality Control District. These included total suspended PM and inhalable PM ( $<10-15 \mu\text{m}$ ), and were averaged to correspond to the sampling periods of the indoor samples. The outdoor samples were supplemented within each geographic cluster using the indoor sampler placed on a stand to protect the pumping-control unit and to hold the sampling inlet at approximately two meters above the ground.

#### Symptoms diaries and peak expiratory flow rate

Symptoms diaries and peak expiratory flow rate (PEFR) measurements were completed by household members over age five for two or three consecutive weeks. Diary symptoms were grouped into three "conditions" for subsequent analyses. These groupings were: 1) symptoms of allergic or irritant responses, such as eye irritation and rhinitis; 2) acute respiratory illness symptoms, including sore throat, cough, wheezing or whistling in the chest, shortness of breath with wheezing, and chest tightness or asthma; and 3) non-specific types of complaints, such as dryness in the mouth, dizziness, fatigue/achy feeling, nausea,

and headache. The occurrence of a symptom on any day implied that the condition was present for that subject. Prevalence rates (per 100 person-days) were calculated and expressed for the period of observation per subject and labeled "period prevalence rates." They required no weighting for minimal differences in reporting periods.

PEFR was measured two to four times per day for at least five days during each testing week using a mini-Wright peak flow meter (Lebowitz 1984; Lebowitz et al. 1985). For each individual, PEFR was analyzed using a two-way analysis of variance (ANOVA) to evaluate the significance of day-of-week (daily) and time-of-day (diurnal) effects. Thus, if an individual's values showed sufficient variability in diurnal or daily PEFRs to yield a statistically significant ANOVA, the individual was classified as "responsive." This provides one statistical test of intraindividual variability and is intended to help classify individuals relative to their likely bronchial responsiveness or susceptibility to air-pollutant exposures.

Data handling and processing procedures for coding and entering data from the questionnaires, sampling, and laboratory data forms were performed on personal computers using the R:Base (Microrim, Inc.) data-base management programs. The data entry procedures were subject to 100% verification using specially designed programs to work with the data base system. Edit checks (limit and consistency) and univariate and bivariate distributional analyses were made to identify incorrect values (e.g., incorrect discrete codes, outliers, range errors) for verification, correction, or identification (flagging). Data transformations and analyses were performed using the SPSS/PC+ statistical programs (SPSS Inc., Chicago, IL), including the Advanced Statistics module for step-wise, log-linear model analyses of categorical data. Standardized prevalence rates for the symptoms and PEFR-responsiveness classifications were derived from the estimated frequencies in the log-linear model (Bishop et al. 1975).

## RESULTS AND DISCUSSION

Results for the summer of 1986 through the spring of 1987 were used for preparation of this summary; 152 samples of  $\text{PM}_{10}$  and  $\text{PM}_{2.5}$  were collected from inside 98 homes and had both laboratory and field records available. Limit checks on the flow rates and filter weights reduced the number of samples to 143 for  $\text{PM}_{10}$  and 141 for  $\text{PM}_{2.5}$ . Consistency checks indicated that two homes with tobacco smoking during the sampling had excessively low values; these were also excluded from the analyses described below. The smok-

2023380324

ing classifications used below were derived from the activity questionnaires corresponding to the sampling periods.

#### *Indoor PM<sub>10</sub> and PM<sub>2.5</sub> concentrations*

Indoor PM<sub>10</sub> and PM<sub>2.5</sub> concentrations were highly correlated as shown in Fig. 1, with an R<sup>2</sup> of 88.6% (n=104 homes). The slope of PM<sub>10</sub> to PM<sub>2.5</sub> was 1.08 with an intercept of 12.5  $\mu\text{g}/\text{m}^3$ ; the mean paired difference was 15 (S.E. 1.2)  $\mu\text{g}/\text{m}^3$ . The slope was slightly higher in homes without smoking (1.16) than in those with smoking (1.07), while the intercept remained fairly constant. This suggests that a larger proportion of the particulate matter in homes without smoking exceeds 2.5  $\mu\text{m}$ . The 12.5  $\mu\text{g}/\text{m}^3$  offset (intercept) represents the overall difference between these PM size cuts and might represent the background contribution to PM<sub>10</sub> from resuspension. Samples collected during the first sampling period generally corresponded with those collected during the following week(s), with an overall R<sup>2</sup> of 85% for both PM<sub>2.5</sub> and PM<sub>10</sub>; the mean paired difference was about 3 (S.E. 2)  $\mu\text{g}/\text{m}^3$ , which was not significantly different than zero ( $p=0.1$ , n=62 and 64 for PM<sub>2.5</sub> and PM<sub>10</sub>, respectively). This suggests that only limited additional information is provided by sampling the home twice during the same season, where both sam-

ples are valid. There were cases, however, when week-specific events (e.g., visitors or fireplace usage) made differences greater than the overall trend. These differences need to be further evaluated in relation to activities, especially nonsmoking-related sources and possible removal mechanisms.

#### *Indoor/outdoor (I/O) PM<sub>10</sub>*

Indoor/outdoor (I/O) PM<sub>10</sub> comparisons were based on inhalable PM measurements made within the same geographic cluster as the home (Fig. 2). In homes without smoking, the median I/O ratio was 0.63 (range 0.3-3.4); the mean paired difference of -4 (S.E. 5.1)  $\mu\text{g}/\text{m}^3$  was not significantly different than zero. In the smoking households, the median I/O ratio was 1.2 (range 0.3-4.7), and the mean difference of 13.5 (S.E. 4.7)  $\mu\text{g}/\text{m}^3$  was significant ( $p<0.01$ ). This indicates that outdoor measurements alone would generally overpredict indoor exposures in nonsmoking households and underpredict those for most homes with tobacco smoking. Indoor/outdoor ratios are highly dependent on indoor activity levels, especially those related to the generation and resuspension of PM, as well as removal rates through ventilation and/or infiltration (Traynor et al. 1985). Additional information collected on the weekly activity questionnaire about these factors in the home needs to be included in

PM<sub>10</sub> VS. PM<sub>2.5</sub> by ETS

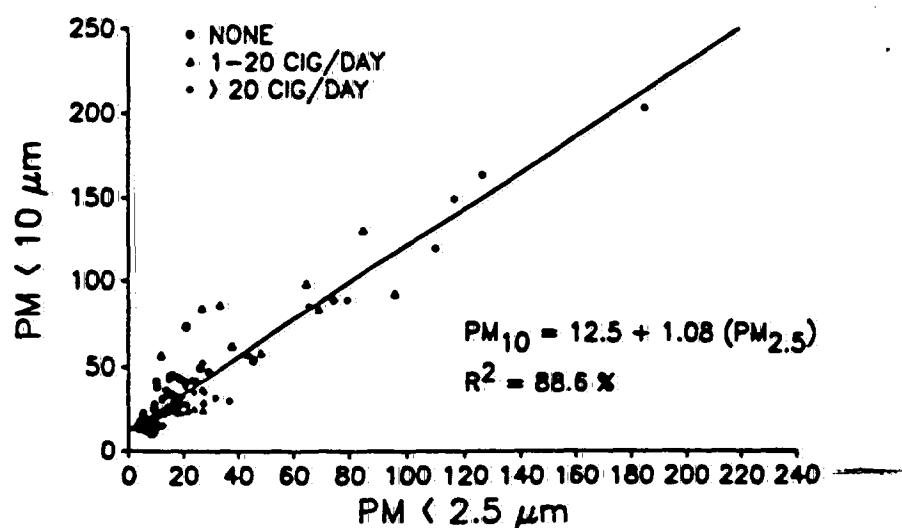


Fig. 1. Comparison of indoor PM<sub>10</sub> with PM<sub>2.5</sub> ( $\mu\text{g}/\text{m}^3$ ) by reported smoking inside the home (cigarettes per day).

2023380325

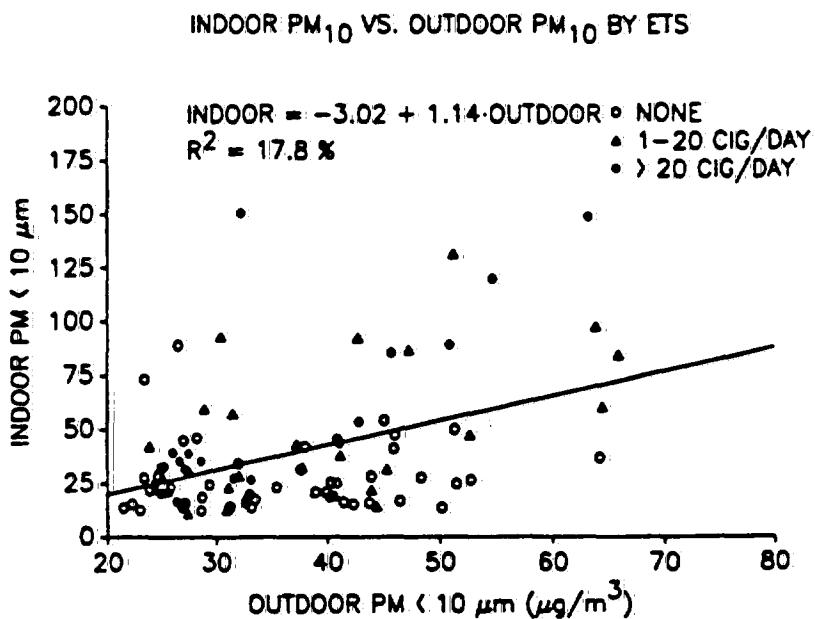


Fig. 2. Comparison of indoor and outdoor PM<sub>10</sub> ( $\mu\text{g}/\text{m}^3$ ) by reported smoking inside the home (cigarettes per day).

subsequent analyses, as well as characterization of the composition of the indoor/outdoor PM.

#### *Measured and estimated PM*

Measured and estimated PM are compared in Table 2. The average PM concentration measured in the home was used for comparison with the estimates of PM from the initial screening questionnaire described above. Overall, the group means are similar to the expected concentrations for respirable suspended PM, although indoor PM<sub>2.5</sub> levels tended to be slightly lower than the estimates. The significance of the ANOVA

supports the ability of the classification methods described here to derive exposure groupings. However, there is considerable variability in measured PM concentrations within each of the classification groups, especially for the medium- and high-exposure classifications. The resulting overlap between groups supports the need to evaluate exposure estimates used in epidemiological studies and to supplement the use of group classifications with direct monitoring, at least in a representative sample of the homes. This will help determine the degree of misclassification error introduced by simplifying assumptions.

Table 2. Comparison of measured PM ( $\mu\text{g}/\text{m}^3$ ) with classification based on reported environmental tobacco smoke.

Estimated	Measured					
	Mean	PM <sub>2.5</sub> S.D.	Homes	Mean	PM <sub>10</sub> S.D.	Homes
≤25	12.9	10.0	(24)	26.0	13.2	(21)
≤40	23.3	21.2	(23)	42.2	27.9	(24)
>40	35.7	39.0	(48)	52.4	43.1	(49)
Total	26.9	31.4	(95)	43.9	36.0	(94)
p(ANOVA)	0.017			0.011		

2023380326

tions (such as those described above) that are used to derive the groupings.

Additional information from the activity questionnaires for the sampling periods was used to help identify sources of misclassification in these groups. Of special concern in this climate is the usage of evaporative ("swamp") coolers in many homes during several months of the year. As shown in Table 3, these act as a significant removal mechanism for different smoking-classification groups. It is possible that they also bring outdoor PM into the home, although these results do not appear support this, since the levels of  $PM_{10}$  and  $PM_{2.5}$  in homes reporting cooler use were about half of the remaining households at all levels of smoking. However, this analysis has not attempted to control for season or outdoor PM levels.

#### $PM_{10}$ exposure and symptoms/PEFR

$PM_{10}$  exposure and symptoms/PEFR classification groups were compared using multinomial statistics and a series of step-wise, log-linear models (controlling for interactions with age and sex). Nonspecific symptoms (i.e. dry mouth, dizziness, fatigue/achy feeling, nausea, or headache) showed a significant ( $p<0.05$ ) interaction with a  $PM_{10}$  classification that was based on the proposed NAAQS of 50 g/m<sup>3</sup>. The adjusted period prevalence rates were 55.9 and 71.2 per 100 subjects for the low ( $\leq 50 \mu\text{g}/\text{m}^3$ ) and high ( $> 50 \mu\text{g}/\text{m}^3$ )  $PM_{10}$  exposure groups respectively. (About 25% of the homes were in the high exposure

group.). These symptoms are similar to those often reported for building-related complaints. They appear to represent annoyance responses to ETS (environmental tobacco smoke) in the higher  $PM_{10}$  group: the average daily prevalence rate was 75% in homes with smoking of more than one pack per day, 52% when up to one pack per day was smoked in the home, and 43% in homes without ETS; these symptoms were primarily in females, who also reported higher prevalences in their children (Lebowitz and Quackenboss 1988). Most of these females, as well as children, were not active smokers. (Active smokers have significantly lower rates of these symptoms.)

The relationship between  $PM_{2.5}$  exposure groups (below or above the median) and symptoms related to acute respiratory illness (ARI) is shown in Table 4 by age and sex groups. The log-linear model for this table consisted of a three-way interaction term relating ARI symptoms to both the PM exposure and age groups and a two-way interaction relating ARI symptoms and sex. The presence of the three-way term indicated that a single age-sex-adjusted prevalence rate should not be calculated for each exposure group and that the relationship between  $PM_{2.5}$  and ARI symptoms is dependent on the individual's age group. This is shown in Table 4, where the trend seems to be reversed for 15-35-year-old women relative to those over 35. Daily prevalence rates of ARI symptoms are related to ETS in homes with lower educational levels (i.e., lower socioeconomic status); the risk ratio

Table 3. Indoor average  $PM_{2.5}$  and  $PM_{10}$  ( $\mu\text{g}/\text{m}^3$ ) by reported smoking in the home and evaporative cooler use during sampling week.

Smoking Cig./d	Evap. Cooler	$PM_{2.5}$			$PM_{10}$		
		Mean	S.D.	Homes	Mean	S.D.	Homes
None	Yes	8.8	5.0	(20)	21.0	9.7	(20)
	No	20.3	19.0	(25)	38.4	22.9	(23)
	Total	15.2	15.5	(45)	30.3	19.9	(43)
1-20	Yes	19.3	8.8	(10)	33.9	12.0	(10)
	No	32.3	28.5	(16)	53.4	33.9	(17)
	Total	27.3	23.6	(26)	46.2	29.1	(27)
>20	Yes	36.2	32.9	(8)	47.4	39.6	(9)
	No	82.7	55.4	(9)	102.5	60.6	(9)
	Total	60.8	50.8	(17)	75.0	57.2	(18)

$PM_{2.5}$ : Significant ( $p<0.01$ ) main effects for smoking and evaporative cooler use; two-way interaction nearly significant ( $p=0.06$ ).

$PM_{10}$ : Significant ( $p<0.01$ ) main effects for evaporative cooler and smoking.

2023380324

Table 4. Prevalence of acute respiratory illness (ARI) symptoms by indoor PM<sub>2.5</sub>, age and sex<sup>a</sup>.

Sex	Age Group (Years)	15 µg/m <sup>3</sup>	15 µg/m <sup>3</sup>	(n)
Male	≤15	41.7	47.4	(43)
	≤35	71.4 <sup>b</sup>	46.2	(20)
	>35	38.5	37.0	(53)
Female	≤15	72.2	82.4 <sup>b</sup>	(35)
	≤35	68.4	41.2	(36)
	>35	30.4	61.1	(59)

<sup>a</sup> Significant ( $p<0.05$ ) 3-way interaction [Age x ARI x PM group] and two-way [Sex x ARI]; unable to calculate age/sex adjusted prevalence rate.

<sup>b</sup> Too few cases with no ARI symptoms for 15-35-year-old males and females 15 years old (n<5 in some cells).

was 2.18 in homes with over one pack per day smoked in the home and 1.64 in those homes with less than one pack per day. There was a relative lack of symptoms in nonsmoking households (25%) in which educational attainment was highest.

Other potentially important factors that might relate to the individual's sensitivity to respiratory illnesses, especially the presence of preexisting respiratory illnesses (e.g., chronic obstructive lung disease, such as chronic bronchitis), will be examined in later papers. ETS per se is not related to these chronic conditions (Lebowitz and Quackenboss 1988), although active smoking is and PM<sub>10</sub> may be. Daily prevalence rates of allergic and irritant symptoms were not re-

lated to ETS, but their incidence rates in relation to outdoor PM need to be evaluated separately.

Intraindividual daily variation in peak flow rates was related to the PM<sub>2.5</sub> grouping, but not to the PM<sub>10</sub> grouping. Age- and sex-adjusted prevalence rates were 31.6% for those exposed to 15 µg/m<sup>3</sup> or less and 45.4% for those with higher indoor PM<sub>2.5</sub> exposures (Table 5). The log-linear model for this table included a significant interaction between PM<sub>2.5</sub> group and daily PEFR responsiveness classification, independent of age and sex. Indoor concentrations of PM<sub>2.5</sub> are related to ETS, as was shown earlier, so that increased variability in PEFR could reflect bronchial responsiveness arising from exposure to ETS.

Table 5. Daily PEFR variability vs. indoor PM<sub>2.5</sub> by age and sex.

Sex	Age Group (Years)	≤15 µg/m <sup>3</sup>	15 µg/m <sup>3</sup>	(n)
Male	≤15	31.6	35.3	(36)
	≤35	14.3 <sup>b</sup>	45.5	(18)
	>35	32.0	61.5	(51)
Female	≤15	37.5	46.7	(31)
	≤35	33.3	43.8	(34)
	>35	31.6	38.2	(53)
Adjusted Prevalence Rate:		31.6	45.4 <sup>b</sup>	(246)

<sup>b</sup> n<5 for unexposed, 15-35-year-old males.

• Significant ( $p<0.05$ ) association between PM<sub>2.5</sub> group and daily variation in PEFR; adjusted rate based on estimates from log-linear model, controlling for age and sex.

2023380328

However, previous findings indicate that bronchial responsiveness was related to PM<sub>2.5</sub> significantly only in homes without ETS (Lebowitz and Quackenboss 1988). On the other hand, diurnal responsiveness was related to ETS in homes with higher PM<sub>10</sub> exposures, after adjusting for age and sex (Lebowitz and Quackenboss 1988). As implied above, PM<sub>10</sub> effects were not independent of ETS. The relationship was strongest in children of both sexes.

The responses to the various indoor pollutants, including PM, may be greater in those with preexisting susceptibility (i.e. physiological, biochemical, and immunological). Further evaluation of responses to PM and ETS will require measurements, such as air nicotine and serum cotinine, that are more specific to ETS. Further evaluation of PM<sub>2.5</sub> speciation is also necessary, since it is a product of several sources.

Day-to-day differences in PEFR could also be related to differences in activity patterns or in other exposures (e.g., occupational, outdoor, or recreational) that will be included in further analyses. These differences could also be related to other pollutants, alone or interactively (Lebowitz 1984; Lebowitz et al. 1985). Some of these interactions may be positive, some negative; passive smoking in children appears to blunt the effects of outdoor ozone, as active smoking does in adults, while children's daily PEFR decrements are enhanced by the interaction of outdoor PM and ozone (Lebowitz 1984; Lebowitz et al. 1985).

The results presented above were based on a number of households (and subjects) in the second stage of our ongoing study. The methods and results illustrate the methodology being used to collect and relate health assessments to exposure measurements. The application of more comprehensive and detailed analyses of the monitoring, questionnaire, symptoms and time budget diary, and PEFR data will be pursued in further papers, and the current results suggest directions for these types of analyses. The relationships between PM exposure and both symptomatic and physiological responses support continued evaluation, especially of individuals who can be identified as more sensitive to the possible health effects of indoor and outdoor air pollutants.

**Acknowledgments** — This work has been supported by EPA Cooperative Agreement No. CR811806 and EPRI Contract No. RP2822. We appreciate the support of the participants and their families, and the field, laboratory, and data-processing staff (Pam Boyer-Pfersdorf, Sue Lanard, Steve Monroe, Mark Sells, and James Winburn), and Kathi Johnson for assistance with SPSS and graphics programs, Donald Burcham of the Pima County Air Quality Control District in obtaining the county's ambient PM data.

The research described in this article has been funded wholly or in part by the U.S. Environmental Protection Agency through Cooperative Agreement No. CR811806. It has not been subjected to the agency's required review and, therefore, does not necessarily reflect the views of the EPA, and no official endorsement should be inferred.

## REFERENCES

- Bishop, Y.M.M., Fienberg, S.W., and Holland, P.W. *Discrete Multivariate Analysis-Theory and Practice*, MIT Press, Cambridge, MA; 1975.
- Dockery, D.W. and Spengler, J.D. Indoor-outdoor relationships of respirable sulfates and particles, *Atmos. Environ.* 15, 335-343; 1981a.
- Dockery, D.W. and Spengler, J.D. Personal exposure to respirable particulates and sulfates, *J. Air Pollut. Control Assoc.* 31, 153-159; 1981b.
- Lebowitz, M.D. The effects of environmental tobacco smoke exposure and gas stoves on daily peak flow rates in asthmatic and non-asthmatic families, *Eur. J. Respir. Dis.* 65 (Suppl. 133), 90-97; 1984.
- Lebowitz, M.D. et al. Respiratory Symptoms and peak flow associated with indoor and outdoor pollutants in the southwest, *J. Air Pollut. Control Assoc.* 35, 1154-1158; 1985.
- Lebowitz, M.D. and Quackenboss, J.J. The effects of environmental tobacco smoke on pulmonary function, *Int. Arch. Occup. and Environ. Health*; 1988.
- Marple, V.A., Rubow, K.L., Turner, W.A. and Spengler, J.D. Low flow rate sharp cut impactors for indoor air sampling: design and calibration, *J. Air Pollut. Control Assoc.* 37, 1303-1307; 1987.
- Quackenboss, J.J., Lebowitz, M.D., and Hayes, C. Epidemiological study of respiratory responses to indoor/outdoor air quality, *Environ. Int.* 15(1-6):493-502; 1989.
- Spengler et al. Long-term measurements of respirable sulfates and particles inside and outside home, *Atmos. Environ.* 15, 23-30; 1981.
- Spengler, J.D. et al. Personal exposures to respirable particulates and implications for air pollution epidemiology, *Environ. Sci. Technol.* 19, 700-707; 1985.
- Traynor, G.W., Nuschke, L.A., and Clarke, W.A. A detailed study of thirty houses with indoor combustion sources (APCA Paper 85-30A.3), *Air Pollut. Control Assoc.*, Pittsburgh, PA; 1985.
- Turner, W.A. and Spengler, J.D. Indoor Aerosol Impactor. Presented at EPA National Symposium on Recent Advances in Measurement of Air Pollutants. Raleigh, NC.; 1985.
- Watson, J.G. and Chow, J.C. The potential of receptor models to apportion indoor air pollution sources (APCA Paper 86-7.6), *Air Pollut. Control Assoc.*, Pittsburgh, PA; 1985.

2023380326

## MEDICAL INTELLIGENCE



## CONCENTRATIONS OF NICOTINE AND TOBACCO SMOKE IN PUBLIC PLACES

WILLIAM C. HINDS, Sc.D.,  
AND MELVIN W. FIRST, Sc.D.

PUBLIC interest has focused on health effects to the large numbers of nonsmokers exposed to tobacco smoke in public places. Recent regulations in a few cities have banned smoking in public places, or have restricted smoking in the manner of United States commercial aircraft.

Two studies<sup>1,2</sup> indicated that in crowded private rooms concentrations of tobacco smoke often exceed 260 µg per cubic meter, the federal air-quality standard for particulate matter that is not to be exceeded more than one day per year. Hoegg<sup>1</sup> estimated that in residences, meeting rooms, or private automobiles, the nonsmoker inhales in one hour the equivalent of smoking 0.01 to 0.20 cigarettes. Bridge and Corn,<sup>2</sup> by measuring carbon monoxide during party situations involving 50 to 73 people in rooms of 140 and 100 m<sup>3</sup> under controlled ventilation conditions, estimated smoke concentrations to be 2000 to 4000 µg per cubic meter and concluded that these levels are a matter of concern.

Estimation of levels of tobacco smoke in public places was undertaken to evaluate the health implications for nonsmokers. Measurements were limited to the particulate phase of tobacco smoke, although it is known that the gaseous phase also contains substances that may affect health. Since the objective was to measure only tobacco smoke, all methods commonly used to measure total suspended particulate matter were ruled out because of the many other sources of particulate matter in the indoor atmosphere. The use of carbon monoxide as a tracer has similar disadvantages because of the widespread distribution of this common air pollutant. Nicotine was chosen as the tracer for tobacco smoke for the following reasons: it is specific for tobacco smoke (the only other source of nicotine is from agricultural sprays, which are unlikely to be a contaminant of the indoor atmospheres tested); with the exception of water, nicotine is the largest single component of the particulate phase of tobacco smoke; nicotine concentration is unaffected by the moisture content of the smoke; and sensitive gas chromatographic analytical methods are available for measurement of nicotine concentrations.

From the Department of Environmental Health Sciences, Harvard School of Public Health (reprint requests should be addressed to Dr. Hinds at the Harvard School of Public Health, 665 Huntington Ave., Boston, MA 02115).

Supported by the Massachusetts Lung Association and its local affiliates.

Because of the wide range of public places evaluated and the small number of samples, the procedures employed and the results should be considered a pilot study having the limited objective of defining the extent of the "passive-smoking" problem in public places.

## SAMPLING METHODS

The procedure was to enter a public place as a patron and sample a known volume of air through an AA Millipore filter having a collection efficiency for tobacco smoke greater than 99 per cent. Samples were taken with an inconspicuous battery-powered pump at a rate of 4 liters per minute for a maximum period of 2½ hours. The entire sampling system weighed 1.3 kg and was contained in a phenolic box, 17 by 19 by 6 cm (Fig. 1). To obtain realistic samples, the unit was placed as close to the breathing zone as possible—e.g., on a table in a restaurant, or on a lap in a train.

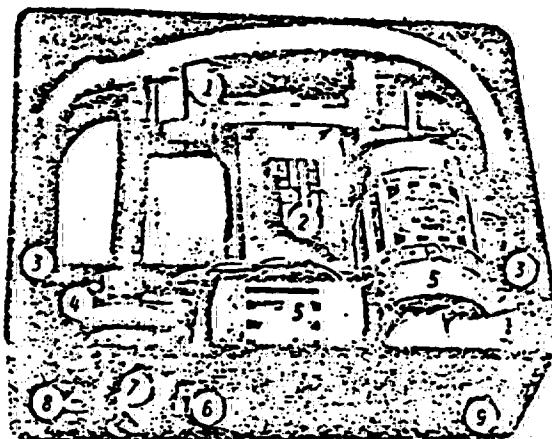


Figure 1. The Sampling System; Showing the Pump (1), Motor (2), Pulsation Damper (3), Filter Holder (4), Rechargeable Batteries (5), On-Off Switch (6), Air Inlet (7), Battery-Charging Jack (8), and Case (9).

The material trapped on the filter was extracted with distilled water, concentrated by rotary evaporation, and analyzed for nicotine with a gas chromatographic technic described by Jacin et al.<sup>3</sup> The nicotine content was used to calculate the tobacco-smoke particulate concentration on the basis of an experimentally determined nicotine fraction of 2.6 per cent established by measurement of total particulate mass and nicotine concentration of sidestream smoke in an aerosol chamber. Sidestream smoke is the principal component of indoor tobacco-smoke pollution (i.e., 80 to 90 per cent).<sup>1,2</sup>

Tests were run with filter and nonfilter cigarettes, and current sales figures<sup>4</sup> were used to calculate the weighted average nicotine fraction as 2.6 per cent. No noteworthy concentration effect on nicotine fraction was observed for smoke concentrations ranging from 6000 to 110,000 µg per cubic meter. Our ambient measurements were an order of magnitude smaller than this range.

Twenty-three samples were taken in the Boston area during 1973 and early 1974. Some types of public areas—commuter trains, commuter buses, and bus and airline waiting rooms—were sampled repeatedly, whereas others, such as large, crowded restaurants and lounges, are represented by individual samples. On buses and trains no attempt was made to sample in smoking or nonsmoking sections because these designations are largely ignored by passengers.

## RESULTS

Smoke concentration for each category of public place is shown in Table 1 as weight per unit volume of sampled

2023380330

**Table 1. Tobacco-Smoke Concentrations in Indoor Public Places.**

CATEGORY	NO. OF SAMPLES	MEASURED NICOTINE CONCENTRATION*	CALCULATED TOBACCO-SMOKE CONCENTRATION*	EQUIVALENT FILTER CIGARETTES SMOKED/Hr.	AVERAGE	RANGE	AVERAGE
					AVERAGE	RANGE	AVERAGE
Commuter train	6	4.9	20-480	190	0.004		
Commuter bus	5	6.3	140-370	240	0.005		
Bus waiting room	2	1.0	16-58	40	0.001		
Airline waiting room	2	3.1	120	120	0.003		
Restaurant	4	5.2	51-450	200	0.004		
Cocktail lounge	3	10.3	170-640	400	0.009		
Student lounge	1	2.8	110	110	0.002		

\* $\mu\text{g}/\text{m}^3$ .

air and "equivalent filter cigarettes per hour," the amount of smoke inhaled by a sedentary nonsmoker in one hour divided by the amount inhaled by a person smoking one filter cigarette (16.1 mg).<sup>1,2,6</sup>

The data on tobacco-smoke concentration presented in Table 1 can be compared to bench marks for clean air based on community ambient-air-quality standards and threshold-limit values for occupational exposures shown in Table 2. These community air-quality standards are based on nontoxic dusts, and it is reasonable to assume that tobacco smoke may be considerably more harmful. The concentrations shown in Table 1 are solely the result of tobacco smoke and do not include the background contribution from usual particulate air pollutants.

The smoke concentrations shown in Table 1 are considerably less than those determined by Hoegg<sup>1</sup> and by Bridge and Corn,<sup>2</sup> who did not account for evaporative losses and diffusive losses to surfaces. Furthermore, calculations based on their data give 12 to 22 per cent of persons smoking at a time and room volumes of 10 to 31 m<sup>3</sup> per person smoking, whereas spot checks made during the present study gave an average of only 9 per cent of people smoking, and room volumes per person smoking ranged from 28 to 4200 m<sup>3</sup>. These differences, at least in part, explain why their calculated concentrations of tobacco smoke are higher by a factor of 10 than our measured values.

The data collected during this study suggest that although tobacco-smoke concentrations often exceed the annual average air quality standard for clean air, these levels would not be expected to produce the strong public reaction to tobacco smoke that has developed in the past few years. This observation suggests that annoyance from

**Table 2. Ambient-Air-Quality Standards and Threshold-Limit Values for Suspended Particulate Matter, Nuisance Dust, and Nicotine.**

SAMPLE	CONCENTRATION
	$\mu\text{g}/\text{m}^3$
Community air-quality standards:	
Suspended particulate matter:	
Annual average	75
Maximum 24-hr concentration (not to be exceeded > once/yr)	260
Occupational standards:	
Nuisance dust:	
Threshold limit value	10,000
Nicotine:	
Threshold limit value	500

tobacco smoke is caused by factors other than the average concentration of particulate matter in the indoor atmosphere. For example, annoyance may be a response to peak concentrations of tobacco smoke that are likely to be much greater than the average values given in Table 1.

Considerable annoyance from tobacco smoking may also result from gaseous components produced during the tobacco combustion. Gaseous components (not including water vapor) represent approximately 70 per cent of the mass of combustion products in sidestream smoke<sup>7</sup> and include strong irritants and unpleasant odors, such as phenols, aldehydes, and organic acids. Awareness of tobacco smoke is enhanced because its submicrometer particle size produces a highly visible aerosol at low mass concentrations. These factors, taken together, may be a more important cause of the public's adverse reaction to tobacco smoke than the quantity measured in the present study, the average smoke concentration.

## REFERENCES

1. Hoegg UR: Cigarette smoke in closed spaces. Environ Health Perspect 2:117-128, 1972
2. Bridge DP, Corn N: Contribution to the assessment of exposure of nonsmokers to air pollution from cigarette and cigar smoke in occupied spaces. Environ Res 5:192-209, 1972
3. Jacin H, Slanski JM, Moshy RJ: The determination of nicotine in tobacco and in particulate matter of smoke by gas chromatography. Anal Chim Acta 41:347-353, 1968
4. Maxwell JC: Leader of the pack. Barrons Weekly, October 29, 1973
5. Wartman WB Jr, Cogbill EC, Harlow ES: Determination of particulate matter in concentrated aerosols: application to analysis of cigarette smoke. Anal Chem 31:1705-1709, 1959
6. Hoffmann D, Wynder EL: Smoke of cigarettes and little cigars: an analytical comparison. Science 178:1197-1199, 1972
7. Scassellati-Sforzolini G, Sabino A: Evaluation of a rapid index of ambient contamination by cigarette smoke, in relation to the composition of the gas phases of the smoke. Riv Ital Igiene 28:43-55, 1968

2023380331

NOTE: THIS IS NOT A  
CERTIFIED TRANSLATION

Annales pharmaceutiques francaises  
36(9-10):443-452, 1978.

ATMOSPHERIC POLLUTION BY SMOKING

by

R. Badre, R. Guillerm, N. Abran, M. Bourdin,  
and C. Dumas

Technical Research Center for Submarine Studies  
(Centre d'Etudes et de Recherches techniques sous-marines)  
83800 Toulon-Naval.

Manuscript received: September 13, 1977.  
Reprints: R. Guillerm (address above).

2023380332

# ATMOSPHERIC POLLUTION BY SMOKING

by

R. Badre, R. Guillerm, N. Abran, M. Bourdin,

and C. Dumas

Technical Research Center for Submarine Studies

(Centre d'Etudes et de Recherches techniques sous-marines)

83800 Toulon-Naval.

## Atmospheric pollution by smoking.

R. BADRE, R. GUILLERM, N. ABRAN, M. BOURDIN and C. DUMAS, Ann. Pharm. Fr., 1978, 36, 443-452.

**SUMMARY.** — After having developed the corresponding analytical and sampling techniques the Authors made in various public gathering places a survey of the respective concentrations in Nicotine, Carbon Monoxide and irritating Pollutants of possible smoke origin.

Nicotine whose unquestionable source is tobacco smoke, was never found in sufficient amount to be harmful for non smokers. Otherwise the concentrations of Carbon monoxide and other non specific pollutants were always found lower than the usually allowed levels and may be considered as innocuous on the toxicological point of view.

However the nuisance which smoke means for non smokers and specially for allergic people and children justifies the restrictive measures usually taken in public places.

## INTRODUCTION

While the general public is becoming more aware of the problems created by atmospheric pollution, numerous public health specialists have been investigating the effects of tobacco smoke on the health of nonsmokers and specifically on the health of children. This pollution by smoking, which is especially noticeable in certain poorly ventilated or crowded public places, has been observed sometimes even in private homes.

Certain authors have described the unfortunate nonsmokers who are obliged to live in an atmosphere polluted by smokers as "passive smokers", and have even issued staunch warnings by declaring that the situation is indisputably dangerous (Hess, 1969 - Cole, 1973 - Dukelow, 1973 - Naumann, 1973 (12,4,15,17)). An important group study was published in 1974 under the direction of RYLANDER (18) (1974), which thoroughly examined the details of the question.

2023380333

It should be noted, however, that not very much precise data on the actual significance of this pollution is available. The procedures used by most authors to determine the concentrations of pollutants created by smoking are subject to criticism: some authors have calculated the theoretical concentrations using the maximal concentrations found in tobacco smoke (HESS, NAUMANN) (12,17); others have worked with entirely artificial situations, such as the accelerated smoking of large quantities of cigarettes in an unventilated area (HARSEN, 1957 (14), SCASSELATI, 1968 cited by DUKELOW (20), COLE, etc.). Still others have calculated concentrations using an element of a different nature to play the role of a tracer, but this is a risky procedure, at best (the particulate phase, for example, was calculated from the carbon monoxide level) (BRIDGE, 1972) (2). Although other serious studies have been carried out, they have mainly dealt with tests performed in the laboratory (HARKE (9,10), 1970 and 1972) and it might be difficult to extrapolate them to concrete real life situations.

An interesting study, with an assortment of samples, was carried out by the American Federal Aviation Administration during 26 airplane trips. Although measurements were only taken of carbon monoxide and the total particulate phase, we will examine the conclusions of this study in the discussion of our results.

We felt that it would be interesting to undertake a study on this topic by discreetly collecting samples in various locations where people smoke, such as cafes, train compartments, and automobiles, and afterwards, to measure the constituents of the pollution, probably originating from smoking, as thoroughly as possible. The first measurements were taken in 1974, but the difficulties which we encountered in using the technique, especially with nicotine, caused us to delay the publication. A relatively recent article by HINDS (1975) (13) revealed that this author has carried out a similar study in Boston, and we will discuss his results later on.

#### THE NATURE OF POLLUTION BY SMOKING

#### THE METHODOLOGICAL APPROACH

Tobacco smoke is a complex aerosol with a composition which varies according to the stream under examination, whether it is the *mainstream smoke*, i.e. the portion which is inhaled by the mouth, or the *sidestream smoke*, i.e., the product of the spontaneous combustion of the cigarette in the air between puffs.

20  
23380334

Atmospheric pollution caused by smoking is essentially due to sidestream smoke. In fact, most of the substances contained in inhaled smoke, which are important from the toxicological point of view, are retained by the organism.

Smoke contains a visible portion, the particulate phase, which is a dense aerosol containing approximately  $10^4$  particles per ml, between 0.1 and 0.8 microns in diameter (C. KEITH and J. DERRICK (16), 1960). The residual gas in air and the gases and vapors resulting from combustion form the gas phase. The complete separation of these two phases is a delicate procedure and, in practice, it is acceptable to consider the gas phase as the product of the filtration of smoke through the Cambridge filter (according to the CORESTA standards).

Although numerous studies have been carried out on the composition of the mainstream, less is known about the sidestream. However, the published studies all agree on one point: qualitatively speaking, the composition of the sidestream is analogous to that of the mainstream. Consequently, the factors considered to be harmful from the toxicological point of view are identical. There is general agreement on the fact that four groups are involved (GUILLERM, 1969, GUILLERM, BADRE et al., 1972) (7,8):

- 1) the alkaloid group, which is essentially nicotine;
- 2) carbon monoxide which interferes with oxygen transport to tissues;
- 3) the group of substances which irritates the respiratory mucosa, is found in the particulate phase as well as in the gas phase, and is essentially composed of aldehydes, ketones, and acids, all of which are water soluble;
- 4) the group of polycyclic hydrocarbons and various substances which are known to be carcinogenic in industrial toxicology.

In the case of atmospheric pollution caused by tobacco smoke, the first three chemical groups are the most relevant:

Nicotine, because of the specific character of its origin, should be a good indicator of pollution from smoking.

Carbon monoxide, which is found in urban pollution, is also important, but it is only a good indicator when other sources are not present.

Irritating substances found in the gas phase, particularly the aldehydes and acrolein, are important, and it is possible to measure them.

As for the polycyclic hydrocarbons, since their infinitesimal levels are already difficult to analyze in the laboratory under optimal conditions, analysis in the field is not very feasible.

2023380335

Because of the reasons mentioned earlier, we have used the following substances as indicators of atmospheric pollution caused by smoking: nicotine, carbon monoxide, and the principal irritating agents of the gas phase.

#### THE ANALYTICAL METHODS

##### 1. The Sample Collection Apparatus

In order to perform our study in the various public places chosen, it was necessary to create an inconspicuous sampling system, which could be contained in a plain valise and could be started easily without having to open the valise. Samples of nicotine, gas needed to measure the carbon monoxide, and traces of pollutants were simultaneously collected on a suitable trap.

a) The sampling device for nicotine was basically composed of "Cambridge" filters placed over the intake of a pump with a known flow rate. Usually, when smoke is aspirated, all of the nicotine, which is almost totally in the particulate phase, can be collected on a "Cambridge" filter. Thus, we thought that we could use these filters to measure the amount of nicotine in smoke present in the air. The low values we obtained, even in a very smoke-filled area, quickly cast doubts on the validity of this technique.

In examining the physical properties of nicotine, we found that its volatile properties were far from being negligible (vapor pressure of pure nicotine at an ambient temperature is close to 0.1 torr, which in saturated vapor corresponds to 1 mg per liter of air). To confirm the risks of loss via this mechanism, we did the following: we impregnated each of two identical Cambridge filters with an equal volume of tobacco smoke condensate solution containing 130 µg of nicotine. We then passed 200 liters of air through one of the filters under the same conditions as for the smoke sampling, i.e. at a rate of 4 l/min. The nicotine was then extracted with cold ethyl acetate and was measured by chromatography. We recovered 107 µg from the control filter and 19 µg from the other filter, thus noting an 80% loss of nicotine. These results confirmed that, although retention of the particulate phase was total, a portion of the nicotine vaporized from the passage of air aspirated through the filter, and also, that a large portion of the nicotine from the particulate phase entered the gas phase when the smoke was dispersed into the air (which further depleted the particulate phase). Thus, it was necessary to find another sampling method which would assure the retention of nicotine in a nonvolatile form, whether it was in the particulate phase or in the vapor phase. We started by employing

2023380336

a technique we had used earlier with CANO (3) (1970), which required water acidified with sulfuric acid, but after a series of tests, we substituted the water for a solution of 1% oxalic acid in 95% ethyl alcohol, since the alcohol promoted the destruction of the smoke aerosol (the principle of the water pipe illustrates the stability of this aerosol when bubbled through water). Under these conditions, after placing three similar scrubbers in a series and passing 200 liters of smoke-filled air through them, we recovered 77% of the total nicotine in the first, 13% in the second, and 10% in the third.

In another test, we added 200 µg of nicotine to the acidic alcohol of the 1st scrubber, passed 400 liters of air through all scrubbers, and measured the nicotine content of each scrubber. We found 171 µg, 4.8 µg, and 1.4 µg, respectively. These results confirmed the presence of a mechanical drive caused by high intensity bubbling, and have enabled us to estimate a maximum of a 5% error in our samples due to a more modest flow rate of 4 l/min. Thus, the final apparatus contained a series of three scrubbers, each containing 15 ml of an acidic alcohol solution, through which 200 liters of air was pumped in 50 minutes (an activated charcoal filter was placed over the exhaust to prevent air pollution by alcohol vapors).

b) The pollutants were also collected by a technique which was developed by us and which we are using at present. It entailed aspirating 100 ml of air through a glass tube filled with a chromatographic adsorbant, Porapak Q, using a syringe whose piston was operated by an electrical motor. The pollutants collected from the volume of air were eluted in the laboratory and were analyzed by gas phase chromatography (BOURDIN, BADRE and DUMAS: 1975) (1).

c) The samples for carbon monoxide analysis were collected by using a pump which filled a 5 liter balloon in 20 minutes (it was previously determined that the balloon was practically impervious to CO for a storage period of less than 24 hours).

## 2. Analytical Techniques

a) Carbon monoxide measurement. The simplest procedure, which is currently used in the area of air pollution, is infrared analysis by the nondispersive method. CO is known to have a specific band which is quite distinct from the CO<sub>2</sub> band. The only element which could interfere in the appropriate zone of sensitivity is water vapor. This interference could be eliminated by drying the gas, or, preferably, by specially arranging receiving chambers to considerably improve the selectivity ("UNOR" apparatus of the Society MAHIAK). We used the apparatus employing the latter method (a scale of 0 to 100 ppm, the limit of detection is on the order of 1 ppm). A sample of a few deciliters was sufficient, and the analysis was instantaneous. The apparatus was recalibrated before testing each sample using a standard mixture of CO in nitrogen.

202338033

b) Measurement of the irritating pollutants. The sample tube was eluted in the laboratory with heat under a stream of nitrogen. The eluted products, collected in a loop immersed in liquid nitrogen, were reheated and injected into the chromatograph only once. This method produced a concentration factor of 100, which enabled us to obtain a great sensitivity. We have already described the methods of operation elsewhere (BOURDIN, BADRE, DUMAS, 1975).

c) Measurement of nicotine. This was carried out by gas phase chromatography using a technique derived from the one described by CANO (3) (1970), except the flame ionization detector was replaced by a thermoionic detector (\*). The separation was performed on a 1/8" column, 1.3 m in length, with the phase being composed of "GASCHROM Q" impregnated with "UCON POLAR 50 HB" 3.2%, and 6% KOH.

The chromatograph was a "Hewlett-Packard" 5750 model with a thermoionic detector specific for nitrogen molecules under the following conditions of operation:

Helium vector gas flow 30 ml/min.

Hydrogen flow 28 ml/min              air 140 ml/min.

Column temperature 110°C.

Detector temperature 400°C.

The acidic alcohol solution from the scrubbers was completely evaporated at a moderate temperature, rediluted in 1N NaOH and extracted twice in 2 ml of ethyl acetate. The extracts were dried on anhydrous  $\text{Na}_2\text{SO}_4$  and were collected in a calibrated, stoppered flask, which enabled calculation of the solvent volume from its mass, since it was so volatile. We then injected 1 to 2  $\mu\text{l}$  into the chromatograph, and calculated the concentration.

#### RESULTS OBTAINED

The results are summarized in table I.

*Carbon monoxide.* The values found were generally very low. In the case of cafe L.R., we found that the inside measured value (23 ppm) was on the same order as the value measured outside on the sidewalk (obvious automobile pollution).

(\*) This provided a significant improvement, since we were able to avoid concentrating the samples because of the sensitivity which was clearly greater to that of the flame ionization. Also, the specificity assured better separation and minimized the solvent "lag".

2023380338

TABLE I. RESULTS OF VARIOUS SAMPLES

15 mars Automail (A)	Compart. train 18 mars (B)	Café G. 26 mars (18 h) (C)	Café I.R. 2 avril 17 h 55 (D)	Café Itv 4 avril 18 h 30 (E)	Café Rg 16 avril 17 h 15 (F)	Café S.i 6 mai 18 h 1 (G)	Hall gare 10 mai 13 h 34 (H)	Café M 15 mai 20 h (I)	Foyer hôpital 15 mai (J)	Compart. train 16 mai (K)	Wagon Pulmann 20 mai (L)	Automobile 30 mai vitre entrouverte (M)	Automob. 30 mai vitre ouverte (N)	Automob. vitres fermées (O)	Chambre étanche (P)
(1) 0,392	3,06	0,64	0,02	0,09	0,48	0,17	3,08	0,14	0,03	0,01	0,34	0	0	2,50	1,64
(2) 0,065	1,01	0,49	0,48	0,17	0,63	2,32	1,41	0,54	0,29	0,38	0,70	0,48	0,26	0,58	
(3) 0,073	0,12	0,05	0,10	0,02	0,15	0,27	0,15	0,07	0,01	0,04	0,10	0,04	0,01	1,00	0,58
(4) 0,07	0,12	0,05	0,07		0,09	0,10	0,04	0,03	0,02	0,02	0,07	0,02	0,03	0,30	0,185
(5) 2,17	0,75	1,12	0,91	5,88	1,31	0,91	0,77	1,40	1,16	0,36	0,67	0,40	0,32	1,20	0,510
(6) 0,46	4,64	2,51	2,07	1,33	3,56	1,47	0,93	1,05	0,82	0,21	1,39	0,14	0,43	1,00	0,40
(7) 0,035	0,10	0,10	0,08	0,15	0,10	0,12	0,05	0,05		0,02	3,17	0,02	0,04	0,15	0,109
(8) 0,119	0,18	0,16	0,15		0,21	0,12	0,09	0,21	0,24	0,09	0,19	0,03	0,03	0,50	0,30
(9) 1,213	2,46	0,69	0,42	0,30	0,54	0,54	0,47	0,37	0,30	0,24	1,03	0,20	0,22	0,30	
(10) 1,41	6,55	5,31	3,00	1,64	2,17	1,27	1,27	0,88	0,73	0,17	1,10	0,34	0,30		
(11) 0,95	0,19	0,38	0,12		0,28										
(12) 0,74	1,87	0,93	0,17	0,04	1,04										
(13) 2,77	5,83	1,28	0,16	0,07	2,59	2,15	3,43	0,42	0,33	0,18	1,71	0,05	0,07	20	0,218
(14) 1	5	11	23	5	14	2	2	7	5	4	2	18	14	0	50
(15)			15	0	7	0	0					0	0		0
(16) 43	50	25	40	30	50	30	20	52	37	36	45	180	65	1010	500
(17) 10/15	2/3	5/16	30/100	10/30	20/60	5/14	20/50	2/8	12/30	2/3	10/20	3/3	3/3	2	18

**KEY:**

- (A) = March 15, Rail car
- (B) = March 18, Train Compart.
- (C) = March 28 (6P.M.) Cafe G
- (D) = April 2 (5:55P.M.) Cafe LR
- (E) = April 4 (6:30 P.M.) Cafe Rv
- (F) = April 16 (5:15 P.M.) Cafe Rg
- (G) = May 6 (6:15 P.M.) Cafe SA
- (H) = May 10 (1:45 P.M.) Train Sta.
- (I) = May 15 (8P.M.) Cafe M
- (J) = May 15 Hospital entrance
- (K) = May 16 Train compart.
- (L) = May 20 Pullman wagon
- (M) = May 30 Auto. window partly open
- (N) = May 30 Auto. window open
- (O) = Auto. windows closed
- (P) = Airtight room

- |                          |                        |
|--------------------------|------------------------|
| (1) Cyclohexane          | (12) Toluene           |
| (2) Ethanal              | (13) 2-Pentanone       |
| (3) Propanal             | (14) CO ppm            |
| (4) Acrolein             | (15) CO exterior       |
| (5) Acetone              | (16) Nicotine          |
| (6) Ethyl acetate        | (17) Number of smokers |
| (7) Benzene              |                        |
| (8) Isopropanol          |                        |
| (9) Ethanol              |                        |
| (10) Methyl ethyl ketone |                        |
| (11) Methyl methacrylate |                        |

Various pollutants. It was difficult to find a correlation between their abundance and the assumed pollution by smoking. They could certainly originate from a great many sources (cooking, heating gas, etc.). Overall, however, the concentrations were very low and were much lower than the maximal allowable concentrations (M.A.C.).

Nicotine. The concentrations found, except in two exceptional cases of an airtight room and a completely closed automobile (\*), were most often lower than or close to  $50 \mu\text{g}/\text{m}^3$  (this theoretically corresponds to the smoke from one cigarette dispersed in a  $20 \text{ m}^3$  room).

#### DISCUSSION

The results obtained for carbon monoxide and for trace pollutants can only be used as indicators because of the nonspecific character of these substances and the diversity of the possible sources. Nevertheless, we can conclude that the concentrations measured at the various test points were considerably lower than the allowable levels (\*\*). Based on these results, which seem to correlate well with the nicotine measurements, it could be stated that the compounds originating from smoking only contribute a small amount to the general air pollution. Especially for carbon monoxide, in cases where the source is essentially smoking (no other sources - exterior pollution low), the concentration usually does not reach the allowable value for a continuous stay in confinement (25 ppm for the 90-day MAC), nor even the lowest value of 8.7 ppm determined by the E.P.A. (Environmental Protection Agency).

As for nicotine, the values found were clearly lower than those determined by authors using theoretical studies or laboratory measurements. On the other hand, they were higher than the values found by HINDS(1975) (13), who measured material trapped on a filter. We have seen that this method contains a basic error, and we have verified, for example, that in the same smoke-filled atmosphere,  $215 \mu\text{g}/\text{m}^3$  was collected by our method of sampling, as opposed to  $14 \mu\text{g}$  with the "Cambridge" filter (32%) and only  $5.5 \mu\text{g}$  (13%) with the "AA Millipore" filter used by HINDS.

(\*) Where the air was almost not breathable.

(\*\*) N.B.: The allowable concentrations in this case are not the allowable values of industrial hygiene for work locations, but much lower concentrations, authorized either by organizations fighting pollution (Environmental Protection Agency) or for a long duration confinement (90-day MAC in a submarine on patrol).

2023380340

This further corresponds to the relationship between our results and the results of Hinds, whose values ranged between 1 and  $10.8 \mu\text{g}/\text{m}^3$ .

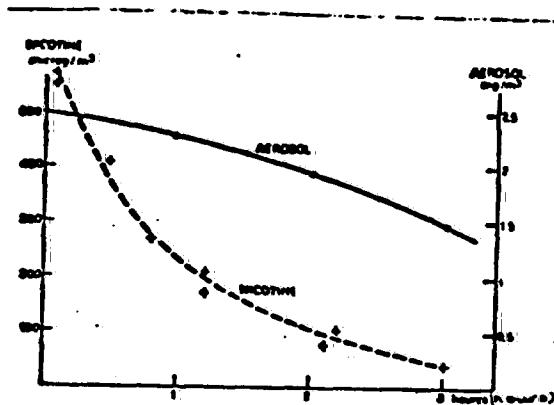


Fig.1. Simultaneous changes of photometric density of smoke and the concentration of total nicotine.

Another confirmation of nicotine depletion in the particulate phase was made by a study, in an enclosed area, of the simultaneous changes in the smoke density (aerosol photometry) and the concentration in total nicotine (fig.1). It was found that the latter decreased much more rapidly, probably through condensation on the walls and ground, which could be demonstrated by measuring, as we did, the nicotine deposited on glass plates in these areas. For example, in 50 minutes, the nicotine level decreased by 50% (half-life), whereas the density of the aerosol decreased by less than 50% in 3 hours. These results were similar to the ones mentioned by MORTON CORN (18) (RYLANDER, ed., 1975) according to which the half-life of the particulate phase was 43 minutes in an airtight room with circulation (curiously, he mentioned 84 minutes for carbon monoxide, which indicates that the room was not air-tight for this gas). In the same study, RYLANDER referred to our earlier results with CANO (3) (1970) by noting that the respective levels of nicotine ( $32 \mu\text{g}/\text{m}^3$ ) and CO (40 ppm) which we had measured were very different from those of ANDERSON and DALHAMN(1973) who found  $0.377 \text{ mg}/\text{m}^3$  and 5 ppm, and from those of HARKE (10)(1972) who found  $0.51 \text{ mg}/\text{m}^3$  and 64 ppm.

It should be noted that we obtained concentrations of  $0.517 \text{ mg}/\text{m}^3$  of nicotine and 50 ppm of CO in our measurements in an airtight room after the smoking of 18 cigarettes per  $40 \text{ m}^3$ , and  $0.05 \text{ mg}/\text{m}^3$  and 5.6 ppm in an ordinary, closed, unventilated

2023380341

room, which confirms the results of Harke and, in general, the values found in short duration tests. In our earlier tests with CANO, we were dealing with very long duration tests (several days). According to other data on the spontaneous decrease in nicotine concentration, it was normal for the mean level to remain permanently quite low while the CO was accumulating (it was even periodically necessary to use a catalytic absorber).

The fact that only low levels of nicotine enter through the airways of "passive smokers" has been confirmed by the determination of nicotine quantities excreted in the urine. In our earlier study with CANO (1970), we demonstrated that non-smoking subjects spending 24 continuous hours in an environment polluted by smokers, with a mean air level of 30 to 40  $\mu\text{g}/\text{m}^3$  of nicotine, eliminated approximately 30  $\mu\text{g}$  of nicotine in urine per 24 hours, while smokers eliminated approximately 1 mg per day. The results have been confirmed by a recent study by RUSSEL and FEYERABEND (19) (1975), which found that urinary levels were approximately 200 times lower in passive smokers as compared to those of smokers.

There seems to be adequate evidence, according to the studies reviewed by RYLANDER (1974), that the respiratory effects of smoke do not lead to significant differences in the rate of respiratory infections in normal nonsmokers. With respect to children, COLLEY (22)(1974) has reported the results of an epidemiological study carried out in over two thousand schoolchildren and their parents. At first, the frequency of a cough in the children appeared to be associated with the smoking habits of their parents. A direct correlation existed between the respiratory symptoms of the parents and those of the children, and when the respiratory symptoms of the parents were taken into consideration, the exposure of the children to cigarette smoke produced by their parents had little effect on the children's symptoms. Thus, the association between the smoking habits of the parents and the respiratory effects in the children that have been reported by other authors and have been interpreted as linked to pollution by smoking, are, in fact, mostly connected with the respiratory diseases of the parents. It is obvious, however, that persons who care for young children, must be dissuaded from smoking in rooms where the children are present.

On the other hand, we cannot deny that certain hypersensitive subjects are inconvenienced by smoke. It has not been possible to clearly demonstrate the allergenic effects of smoke (RYLANDER, 1974). However, there is no doubt that a large proportion of nonsmokers exposed to smoke experiences eye irritation, at least, and a study dealing with this matter was carried out in 1971 during air travel by the

2023380345

Federal Aviation Administration and the N.I.O.S.M. in the U.S.A. (6). The questionnaire, given to 3,296 passengers, showed that, despite the low level of pollution indicated by the CO measurements, 60% of the nonsmokers, and even 20% of the smokers, were annoyed by the smoking of other passengers. Finally, it should be noted that numerous asthmatics are bothered by smoke-filled environments, through a mechanism which requires more thorough investigation; it is not caused by an allergenic type of mechanism, but by an increase in the nonspecific sensitivity of the bronchial receptors to the irritants in smoke or by a psychosomatic phenomenon.

#### CONCLUSION

The measurements, which we performed under realistic conditions, showed that the concentrations of carbon monoxide and the nonspecific compounds of tobacco smoke in the air of places where there was smoking were not increased sufficiently to create a toxicological risk for nonsmokers sharing areas with smokers.

As for nicotine, the total concentration in air polluted by tobacco smoke was often low and was usually below  $50 \mu\text{g}/\text{m}^3$ . If we were to assume, for example, that the nonsmoker spent 10 hours per day (work and travel) in  $50 \mu\text{g}/\text{m}^3$  smoke-filled air, he would inhale 400  $\mu\text{g}$  of nicotine, of which he would retain 80%, at most, or 320  $\mu\text{g}$ . When compared with what the smoker inhales on the average, i.e. around 1  $\mu\text{g}/\text{cigarette}$  it would be the equivalent of the nonsmoker smoking only 40% of one cigarette, while the smoker smoked about twenty. Strictly from the toxicological point of view, we could thus say that smoking does not present a risk to nonsmokers.

Statements which depict the "passive smoker" as a victim whose health is being threatened by his neighbor's smoking habit are truly irrational, particularly because of the psychosomatic incidents, which are especially evident in asthmatics, and have also been known to occur with many nonsmokers. It is equally irresponsible to overlook the fact that smokers will satisfy their tobacco habits anytime and anyplace ignoring the fact that they are annoying a large proportion of the population, as was thoroughly demonstrated by the F.A.A. study. Thus, we are supporting those activities which will reinforce and complement the already existing measures that have been effective but are now being neglected, especially the mass transportation regulations which reserve compartments for nonsmokers.

2023380343

## BIBLIOGRAPHY

- [1] BOURDIN (M.), DUKE (R.) et DUMAS (C.). *Analisis*, 1975, 3, p. 34-38.
- [2] BRIGG (D.P.) et CORN (M.). *Environmental Research*, 1972, 5, p. 192-209.
- [3] CANO (J.P.), CAILLEN (J.), BAUER (R.), DUMAS (C.), VIALA (A.) et GUILLERMI (R.). *Ann. pharm. fr.*, 1970, 28, p. 561-565.
- [4] COLE (P.V.) et BROWNS (E.). *Lancet*, 1973, I, p. 576-579.
- [5] DUKKOW (D.A.). *J.A.M.A.*, 1973, 223, p. 336-337.
- [6] Federal Aviation Administration (anonyme) (1971). *Health aspects of smoking in transport aircraft*. Report U.S. Public Health Service Depart' of Health, Education and Welfare.
- [7] GUILLERMI (R.). *Les Cahiers Sandoz*, 1969, 15, p. 29-47.
- [8] GUILLERMI (R.), BUDRE (R.), HEE (J.) et MASUREL (G.). *Revue de Tuberculose et de Pneumologie*, 1972, 36, p. 187-206.
- [9] HARKE (H.P.). *Münch. Med. Wochr.*, 1970, 812, n° 51, p. 2328-2334.
- [10] HARKE (H.P.) et coll. *Int. Arch. Arbeitsmed.*, 1972, 29, p. 323-339.
- [11] HERMSEN (H.) et EFFEKSBERGER (E.). *Arch. Hyg.*, 1957, 141, p. 383.
- [12] HESS (M.). *Litteratur Eildienst*, Roche, 1959, 37, p. 49.
- [13] HINNS (W.C.) et FIRTS (M.W.). *New England J. Med.*, 1975, p. 844-845.
- [14] HORNING (E.C.) et coll. *Life Sciences*, 1973, 13, p. 1331-1346.
- [15] HUBER (G.). *New England Journal Med.*, 1975, p. 858-859.
- [16] KENT (C.), DERRICK (J.). *J. Coll. Sc.*, 1969, 15, 340-356.
- [17] NAUSCHN (R.J.). *Sciences*, 1973, 182, n° 4110, p. 334-336.
- [18] RALANDER (R.). *Scandinavian Journal of Resp. Diseases*, 1974, Suppl. n° 91.
- [19] RUSSEL (M.A.) et FEYERABEND (C.). *Lancet*, 1975, I, p. 79-181.
- [20] SCARNIATI et coll. *Riv. Ital. Igiene*, 1968, 26, p. 43-55.
- [21] TAYLOR (G.). *Scand. J. Resp. Dis.*, 1974, Suppl. n° 91, p. 50-55.
- [22] COLLEY (J.R.T.). *Brit. med. Journal*, 1974, 2, p. 201-205.

2023380344

## Pollution atmosphérique par la fumée de tabac

R. BADRE, R. GUILLERM, N. ABRAN, M. BOURDIN et C. DUMAS

Centre d'Etudes et de Recherches techniques sous-marines, 83300 Toulon-Naval.

### Atmospheric pollution by smoking.

R. BADRE, R. GUILLERM, N. ABRAN, M. Bourdin and C. Dumas, Ann. Pharm. Fr., 1978, 36, 443-452.

**SUMMARY.** — After having developed the corresponding analytical and sampling technics the Authors made in various public gathering places a survey of the respective concentrations in Nicotine, Carbon Monoxide and Irritating Pollutants of possible smoke origin.

Nicotine whose unquestionable source is tobacco smoke, was never found in sufficient amount to be harmful for non smokers. Otherwise the concentrations of Carbon monoxide and other non specific pollutants were always found lower than the usually allowed levels and may be considered as innocuous on the toxicological point of view.

However the nuisance which smoke means for non smokers and specially for allergic people and children justifies the restrictive measures usually taken in public places.

### Pollution atmosphérique par la fumée de tabac.

R. BADRE, R. GUILLERM, N. ABRAN, M. Bourdin et C. Dumas, Ann. Pharm. Fr., 1978, 36, 443-452.

**Résumé.** — Après avoir mis au point les techniques de prélèvement et d'analyse, les auteurs ont effectué dans divers lieux publics des contrôles portant sur la nicotine, le monoxyde de carbone et les polluants irritants de la fumée de tabac.

La nicotine dont l'origine tabagique est seule indiscutable n'a jamais été retrouvée en quantité assez importante pour qu'elle présente des risques pour les non-fumeurs. De même les concentrations en monoxyde de carbone et en autres polluants non spécifiques sont restées inférieures aux normes habituellement admises et ne présentent donc pas de danger toxicologique. Cependant, la gêne que représente la fumée pour les non-fumeurs et notamment les allergiques et les jeunes enfants justifie les mesures de restriction habituelles dans certains lieux publics.

## INTRODUCTION

En même temps que le grand public prenait conscience des problèmes que pose la pollution de l'atmosphère, de nombreux hygiénistes se sont préoccupés des incidences que pouvait avoir sur la santé des non-fumeurs et singulièrement

Manuscrit reçu le 13 septembre 1977.  
Tirés à Part : R. Guillerm (à l'adresse ci-dessus).

2023380345

des enfants la pollution produite par la fumée de tabac. Cette pollution tabagique particulièrement perceptible dans certains lieux publics mal ventilés ou surpeuplés est également sensible, parfois, jusque dans les logements privés.

Certains auteurs ont assimilé à des « fumeurs passifs » les malheureux non-fumeurs ainsi obligés de vivre dans l'atmosphère polluée par les fumeurs et ils ont même lancé de véritables cris d'alarme en affirmant qu'il s'agissait d'un indiscutable danger (HESS, 1969 — COLE, 1973 — DUKELOW, 1973 — NAUMANN, 1973 [12, 4, 15, 17]). Une remarquable étude réalisée en équipe a été publiée en 1974 sous la direction de RYLANDER [18] (1974) et fait parfaitement le point de la question.

Il convient cependant de préciser qu'on dispose de très peu de données précises sur l'importance réelle de cette pollution. La plupart des auteurs ont déterminé les concentrations des polluants d'origine tabagique par des procédés critiquables : certains ont calculé les concentrations théoriques à partir des concentrations maximales trouvées dans la fumée de tabac (HESS, NAUMANN) [12, 17] ; d'autres se sont placés dans des conditions tout à fait artificielles, tel que le fumage accéléré de très nombreuses cigarettes dans un local non ventilé (HERMSEN, 1957 [14], SCASSELATI, 1968 cité par DUKELOW [20], COLE, etc.). D'autres encore ont calculé les concentrations à partir de celles d'un élément de nature différente, jouant le rôle d'un traceur (par exemple phase particulaire calculée à partir du taux de monoxyde de carbone), ce qui est pour le moins hasardeux (BRIDGE, 1972) [2]. Bien que d'autres travaux aient été faits avec beaucoup de sérieux, ils portent essentiellement sur des essais effectués en laboratoire (HARKE [9, 10], 1970 et 1972) dont on peut penser qu'il est difficile de les extrapolier aux cas concrets de la vie courante.

Une enquête intéressante, assortie de prélèvements, a été effectuée par l'Aéronautique Américaine au cours de 26 voyages aériens. Bien que les dosages n'aient porté que sur le monoxyde de carbone et la phase particulaire totale, nous reviendrons sur les conclusions de cette étude dans la discussion de nos résultats.

Il nous a donc semblé intéressant d'entreprendre une étude sur le terrain en procédant à des prélèvements discrets dans divers lieux où l'on fume tels que salles de café, compartiments de chemin de fer, automobiles, en vue d'effectuer, en différé, des déterminations aussi complètes que possible des éléments de pollution d'origine tabagique probable. Les premières mesures remontent à 1974 mais les difficultés que nous avons rencontrées dans la mise au point de la technique, notamment pour la nicotine, nous ont conduit à en retarder la publication. Un article relativement récent de HINDS (1975) [13] révèle que cet auteur a procédé à une étude similaire dans la région de Boston et nous en discuterons plus loin les résultats.

2023380346

## NATURE DE LA POLLUTION TABAGIQUE APPROCHE METHODOLOGIQUE

La fumée de tabac est un aérosol complexe dont la composition varie selon que l'on a affaire au courant primaire de la fumée, c'est-à-dire à la partie qui est aspirée par la bouche ou au courant secondaire, c'est-à-dire au produit de la combustion spontanée de la cigarette à l'air entre deux bouffées.

Dans le cas de la pollution atmosphérique d'origine tabagique, c'est essentiellement le courant secondaire de la fumée qui est en cause. En effet, la plus grande partie des substances contenues dans la fumée inhalée qui présentent de l'intérêt au plan toxicologique est retenue par l'organisme.

La fumée comporte une partie visible, la phase particulaire qui est un aérosol dense renfermant par ml environ 10<sup>6</sup> micelles dont le diamètre est compris entre 0,1 et 0,8 microns (KERN C. et DERRICK J. [16], 1960). Les gaz résiduels de l'air et les gaz et vapeurs résultant de la combustion forment la phase gazeuse. La séparation parfaite de ces deux phases est délicate et dans la pratique on a admis que l'on considérerait comme phase gazeuse le produit de la filtration de la fumée sur un filtre Cambridge (selon les normes CORESTA).

Si de très nombreuses recherches ont été effectuées sur la composition du courant primaire, on connaît moins bien celle du courant secondaire. Cependant, les travaux publiés concordent tous sur un point : qualitativement parlant, la composition du courant secondaire est analogue à celle du courant primaire. En conséquence, les facteurs de nuisances à considérer au plan toxicologique sont identiques. On s'accorde généralement à considérer qu'ils appartiennent à quatre groupes (GUILLERU, 1969, GUILLERU, BIROU et coll., 1972) [7, 8] :

- 1) le groupe des alcaloïdes, c'est-à-dire essentiellement la nicotine;
- 2) le monoxyde de carbone dont l'effet s'exerce sur le transport d'oxygène aux tissus;
- 3) le groupe des substances irritantes pour la muqueuse respiratoire réparties aussi bien dans la phase particulaire que dans la phase gazeuse, et qui comprend essentiellement des composés aldéhydiques, cétoniques ou acides, tous hydrosolubles;
- 4) le groupe des hydrocarbures polycycliques et diverses substances qui, en toxicologie industrielle, sont connues pour être cancérogènes.

Dans le cas de la pollution atmosphérique par la fumée de tabac, ce sont les trois premières familles chimiques qui présentent le plus d'intérêt :

Le nicotine, en raison du caractère spécifique de son origine, devrait constituer un bon traceur de la pollution tabagique.

Le monoxyde de carbone, que l'on retrouve dans la pollution urbaine, est également intéressant mais il n'est un bon traceur de la phase gazeuse qu'en l'absence d'autres sources.

Les substances irritantes se trouvant dans la phase gazeuse, notamment les aldéhydes et l'acroléine, sont à considérer et leur dosage est possible.

Quant aux hydrocarbures polycycliques, compte tenu de leur teneur infinitésimale leur analyse déjà difficile au laboratoire dans les meilleures conditions est peu praticable sur le terrain.

Pour les raisons précitées, nous avons donc retenu comme représentatives de la pollution atmosphérique d'origine tabagique les substances suivantes : nicotine, oxyde de carbone, irritants principaux de la phase gazeuse.

2023380347

## METHODES ANALYTIQUES

### I. LE DISPOSITIF DE PRÉLEVEMENT.

Afin de pouvoir opérer dans les divers lieux publics jugés intéressants il a été nécessaire de réaliser un ensemble discret, logé à l'intérieur d'une valise banale et pouvant être mis en route à volonté sans avoir à ouvrir la valise. Il assure simultanément le prélèvement de la nicotine, de l'échantillon gazeux nécessaire au dosage du monoxyde de carbone et celui des polluants à l'état de traces sur un piège approprié.

a) Le dispositif de prélèvement de la nicotine était initialement constitué par des filtres « Cambridge » placés sur l'aspiration d'une pompe de débit connu. Au moment de l'émission de la fumée, la nicotine se retrouve en effet pratiquement en totalité dans la phase particulaire et peut être intégralement récoltée sur filtre « Cambridge ». Nous pensions donc pouvoir utiliser ces mêmes filtres pour doser la nicotine de la fumée dispersée dans l'atmosphère. La faiblesse des valeurs obtenues même dans un local très enflamé nous a rapidement conduits à mettre en doute la validité de cette technique.

Un examen des propriétés physiques de la nicotine permet effectivement de constater que sa volatilité est loin d'être négligeable (la tension de vapeur de la nicotine pure à température ambiante est voisine de 0,1 torr, ce qui correspond en vapeur saturante à 1 mg par litre d'air). Pour confirmer les risques de perte par ce mécanisme nous avons effectué l'essai suivant : deux filtres Cambridge identiques ont été imprégnés chacun d'un même volume d'une solution de condensat de fumée de tabac renfermant 130 µg de nicotine. À travers l'un des 2 filtres on a fait passer 200 litres d'air dans les conditions de prélèvement de la fumée, soit au débit 4 l/mn. Les 2 filtres ont ensuite été épulisés par l'acétate d'éthyle à froid et on a dosé la nicotine par chromatographie. On a trouvé ainsi 107 µg dans le témoin et 19 µg dans l'autre, soit une perte de 80 % de la nicotine. Ces résultats confirment que, même si la rétention de la phase particulaire est totale, une partie de la nicotine se vaporise à la faveur du balayage par l'air aspiré sur le filtre et que, en outre, une partie importante de la nicotine de la phase particulaire passe en phase vapeur lorsque la fumée se répand dans l'atmosphère (ce qui appauvrit encore la phase particulaire). Il a donc été nécessaire de rechercher une autre méthode de prélèvement assurant la rétention de la nicotine sous une forme non volatile, que celle-ci se trouve dans la phase particulaire ou dans la phase vapeur en utilisant d'abord de l'eau acidulée par l'acide sulfurique selon une technique que nous avions utilisée précédemment avec CANO [3] (1970) puis, à la lumière des résultats obtenus sur des essais systématiques, une solution à 1 % d'acide oxalique dans l'alcool éthylique à 95°, l'alcool favorisant la destruction de l'aérosol de fumée (la pratique du « narghilé » illustre la stabilité de cet aérosol au barbotage dans l'eau). Dans ces conditions en plaçant trois barboteurs semblables en série on trouve, après passage d'environ 200 litres d'air enflamé, 77 % de la nicotine totale dans le premier, 13 % dans le second et 10 % dans le troisième.

Dans un autre essai l'alcool acide du 1<sup>er</sup> barboteur étant préalablement additionné de 200 µg de nicotine on a dosé la nicotine dans chacun des barboteurs après passage de 400 litres d'air. On a trouvé respectivement 171-4,8 et 1,4 µg. Ce résultat confirme la réalité d'un entraînement mécanique dû au barbotage à grand débit et permet d'estimer à 5 % au maximum l'erreur par défaut dont sont entachés les prélèvements au débit plus modeste de 4 litres/minutés que nous avons utilisé. Le dispositif finalement retenu comportait donc un train de 3 barboteurs renfermant chacun 15 ml de solution alcoolique acide à travers lesquels une pompe faisait passer 200 litres d'air en 50 minutes (un filtre à charbon actif placé sur le resoufflement évitait de polluer l'atmosphère en vapeur d'alcool).

2023380348

b) Les polluants étaient recueillis par ailleurs selon une technique mise au point et couramment utilisée par nous. Elle consiste en l'emploi d'un tube de verre garni d'un adsorbant chromatographique, le Porapak Q, à travers lequel on aspire 100 ml d'air à l'aide d'une seringue dont le piston est mû par un moteur électrique. Les polluants renfermés dans le volume d'air recueilli sont désorbés en laboratoire et analysés par chromatographie en phase gazeuse (BOURDIN, BADRE et DUMAS, 1975) [1].

c) Le prélèvement destiné à l'analyse du monoxyde de carbone était effectué à l'aide d'une pompe qui en 20 minutes remplit un ballon souple de 5 litres (dont on a vérifié préalablement qu'il était pratiquement imperméable au CO pour une durée de conservation inférieure à 24 heures).

## 2. TECHNIQUES ANALYTIQUES

a) Dosage du monoxyde de carbone Le procédé le plus simple, d'usage courant dans le domaine de la pollution atmosphérique, est l'analyse infrarouge par la méthode non dispersive. On sait que le CO possède une bande spécifique bien distincte de celle du CO<sub>2</sub>. Le seul élément qui peut interférer dans la zone de sensibilité souhaitée est la vapeur d'eau. Cette interférence peut être éliminée par dessiccation du gaz ou mieux encore par une disposition spéciale des chambres réceptrices qui améliore considérablement la sélectivité (appareil « UNOR » de la Société MULLAK). C'est ce dernier appareil que nous avons utilisé (échelle de 0 à 100 ppm, limite de détection de l'ordre de 1 ppm). Un prélèvement de quelques décilitres est suffisant et l'analyse instantanée. L'appareil a été récalibré avant chaque dosage à l'aide d'un mélange étalon de CO dans l'azote.

b) Dosage des polluants irritants. Le tube de prélèvement est désorbé en laboratoire par balayage à chaud sous courant d'azote. Les produits désorbés sont recueillis dans une boucle plongée dans l'azote liquide et après réchauffage injectés en un seul temps dans le chromatographe. Cette opération réalise une concentration par un facteur 100, ce qui permet d'obtenir une grande sensibilité. Le mode opératoire détaillé a été décrit par nous précédemment (BOURDIN, BADRE, DUMAS, 1975).

c) Dosage de la nicotine. Il a été effectué par chromatographie en phase gazeuse par une technique dérivée de celle que nous avons décrite avec CANO [3] (1970) mais où le détecteur à ionisation de flamme était remplacé par un détecteur thermoionique (\*). La séparation est faite sur une colonne de 1/8" et 1,30 m de long, la phase étant constituée par du « GASCHROM Q » imprégné de « UCON POLAR 50 HB » 3,2 % et KOH 6 %.

Le chromatographe est un « Hewlett-Packard » modèle 5750 avec détecteur thermoionique spécifique des molécules azotées dans les conditions opératoires suivantes :

Debit gaz vecteur Hélium 30 ml/mn.  
 ► hydrogène 28 ml/mn      air 140 ml/mn.  
 Température colonne 110 °C.  
 ► détecteur 400 °C.

La solution alcoolique acide provenant des barboteurs est évaporée à sec à température modérée puis reprise par NaOH 1N et extraite en deux fois par 2 ml d'acétate d'éthyle. Les extraits, séchés sur SO<sub>3</sub>Na, anhydre sont recueillis en flacon bouché tari, ce qui permet de calculer le volume de solvant à partir de sa masse en raison de sa volatilité. On injecte 1 à 2 µl dans le chromatographe et calcule la concentration.

(\*) Cela apporte une amélioration importante, la sensibilité étant nettement supérieure à celle de l'ionisation de flamme, ce qui permet d'éviter de concentrer les échantillons. La spécificité assure en outre une meilleure séparation et minimise la « trainée » du solvant.

2023380349

TABLEAU I. — Résultats de

	15 mars Autorail	Compart. train 18 mars	Café G. 26 mars (18 h)	Café L.R. 2 avril. 17 h 55	Café Rv 4 avril 18 h 30	Café Rg 16 avril 17 h 15	Café S 6 m 18 h
Cyclohexane .....	0,392	3,06	0,64	0,02	0,09	0,48	0,17
Ethanal .....	0,065	1,04	0,49	0,48	0,17	0,63	2,32
Propanal .....	0,073	0,12	0,05	0,10	0,02	0,15	0,27
Acroléine .....	0,07	0,12	0,05	0,07		0,09	0,10
Acétone .....	2,17	0,75	1,12	0,91	5,88	1,31	0,51
Acétate d'éthyle ..	0,46	4,64	2,51	2,07	1,33	3,56	1,41
Benzène .....	0,035	0,10	0,10	0,08	0,15	0,10	0,12
Isopropanol ...	0,119	0,18	0,16	0,15		0,21	0,11
Ethanol .....	1,213	2,46	0,69	0,42	0,30	0,54	0,5
Méthyhéthyl- cétone .....	1,44	6,55	5,31	3,00	1,64	2,17	1,2
Méthacrylate de méthyle ....	0,95	0,19	0,38	0,12		0,28	
Toluène .....	0,74	1,87	0,93	0,17	0,04	1,04	
2 Pentanone ...	2,77	5,83	1,28	0,16	0,07	2,59	2,1
ppm {							
CO ppm .....	1	5	11	23	5	14	2
CO extérieur ...				15	0	7	0
μg/m³ {							
Nicotine .....	43	50	25	40	30	50	30
Nombre de fumeurs .....	10/15	2/3	5/16	30/100	10/30	20/60	5/1

## RESULTATS OBTENUS

Ils sont rassemblés dans le tableau I.

**Oxyde de carbone.** Les valeurs trouvées sont en général très faibles. Dans le cas du café L.R. on a contrôlé que la valeur trouvée (23 ppm) était du même ordre que celle qui a été mesurée sur le trottoir extérieur (pollution automobile évidente).

**Polluants divers.** Il est difficile de trouver une corrélation entre leur abondance et la pollution tabagique supposée. Cela résulte certainement du grand nombre de sources possibles (gaz d'échappement, cuisine, etc.). Dans l'ensemble les concentrations sont néanmoins très faibles et très inférieures aux concentrations maximales admissibles (C.N.A.).

**Nicotine.** Les concentrations trouvées, sauf dans les deux cas exceptionnels de la chambre étanche et de la voiture entièrement fermée (\*), sont le plus souvent inférieures ou voisines de 50 μg/m³ (ce qui correspond théoriquement à la fumée d'une cigarette entrouverte dispersée dans une pièce de 20 m³).

(\*) Dont l'atmosphère était presque irrespirable.

2023380350

différents prélèvements.

Hall gare 10 mai 13 h 14	Café M 15 mai 20 h	Foyer hôpital 15 mai	Compart. train 16 mai	Wagon Pulmann 20 mai	Automobile 30 mai vitre entrouverte	Automob. 30 mai vitre ouverte	Automob. vitres fermées	Chambre étanche
3,08	0,14	0,03	0,01	0,34	0	0	2,50	1,64
1,41	0,54	0,29	0,38	0,70	0,48	0,26	1,00	0,58
0,15	0,07	0,01	0,04	0,10	0,04	0,01	0,30	0,185
0,04	0,03	0,02	0,02	0,07	0,02	0,03	0,15	0,09
0,77	1,40	1,16	0,36	0,67	0,40	0,32	1,20	0,510
0,93	1,05	0,52	0,21	1,39	0,14	0,43	1,00	0,40
0,05	0,05		0,02	3,17	0,02	0,04	0,15	0,109
0,09	0,21	0,24	0,09	0,19	0,03	0,03	0,50	0,30
0,47	0,37	0,30	0,24	1,03	0,20	0,22	0,30	
1,27	0,88	0,73	0,17	1,10	0,34	0,30		
				4,61			0,50	0,218
3,43	0,62	0,33	0,18	1,71	0,05	0,07	20	
2	7	5	4	2	18	14	0	50
0					0	0		0
20	52	37	36	45	160	65	1010	500
20/50	2/8	12/30	2/3	10/20	3/3	3/3	2	18

## DISCUSSION

Les résultats obtenus tant pour le monoxyde de carbone que pour les polluants à l'état de traces n'ont qu'une valeur indicative en raison du caractère non spécifique de ces substances et de la diversité des sources possibles. On peut néanmoins conclure que les concentrations mesurées dans les divers points contrôlés restent notablement inférieures aux niveaux admissibles (\*). En se basant sur les résultats qui paraissent en bonne corrélation avec les dosages de nicotine on peut dire que les composés d'origine tabagique ne contribuent que pour une très faible part à la pollution générale de l'atmosphère. En particulier pour le monoxyde de carbone, dans les cas où son origine semble devoir être attribuée essentiellement à la fumée de tabac (pas d'autres sources — pollution extérieure faible), sa concentration n'atteint pas le plus souvent les valeurs admises pour le séjour

(\*) N.B. : Les concentrations considérées comme admissibles dans ce cas ne sont pas les valeurs admises en hygiène industrielle pour les locaux de travail mais les concentrations, beaucoup plus faibles, autorisées soit par les organismes de lutte contre la pollution (Environmental Protection Agency), soit pour le confinement de longue durée (CMA 90 jours des sous-marins en patrouille).

2023380351

continu en confinement (25 ppm pour la CMA 90 jours) et même la valeur très basse de 8,7 ppm fixée par l'E.P.A. (Environmental Protection Agency).

En ce qui concerne la nicotine les valeurs trouvées sont nettement inférieures à celles qui ont été avancées par les auteurs partant soit d'études théoriques, soit de mesures en laboratoire. Elles sont au contraire très supérieures à celles qu'a obtenu Hixson (1975) [13] en utilisant le prélèvement sur filtre. Nous avons vu que cette méthode comporte une erreur de principe et nous avons vérifié par exemple que dans une même atmosphère enflamée on trouvait 215 µg/m<sup>3</sup> par notre méthode de prélèvement contre 14 µg avec filtre « Cambridge » (32 %) et seulement 5,5 µg (13 %) avec le filtre « Millipore AA » utilisé par Hixson. Cela correspond d'ailleurs au rapport de nos résultats et de ceux de cet auteur dont les valeurs s'échelonnent entre 1 et 10,8 µg/m<sup>3</sup>.

Une autre confirmation de l'appauvrissement de la phase particulaire en nicotine est apportée par l'étude en local clos de l'évolution simultanée de la densité de fumée (photométrie de l'aérosol) et de la concentration en nicotine totale (fig. 1). On constate que

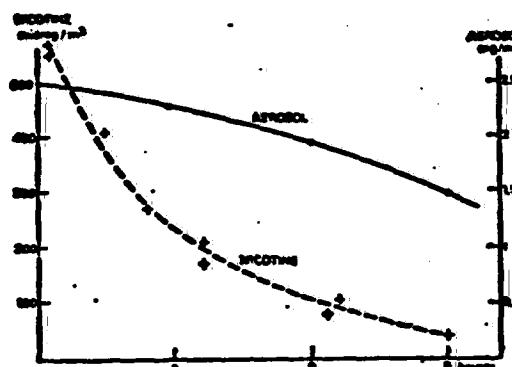


FIG. 1. — Evolution simultanée de la densité photométrique de fumée et de la concentration en nicotine totale.

cette dernière diminue beaucoup plus rapidement, probablement par condensation sur les parois et le sol ainsi qu'on peut le montrer en dosant comme nous l'avons fait la nicotine déposée sur des plaques de verre placées à cet effet. Par exemple en 50 minutes le taux de nicotine diminue de 50 % (demi-vie) alors que la densité d'aérosol baisse de moins de 50 % en 3 heures. Ces résultats sont à rapprocher de ceux que résume Monotox CORX [18] (RYLANDER, édit., 1975) selon lesquels la demi-vie de la phase particulaire serait de 43 minutes en local étanche avec brassage (curieusement il indique 84 minutes pour le monoxyde de carbone, ce qui tendrait à prouver que la chambre n'est pas étanche pour ce gaz). Dans la même étude RYLANDER fait état de nos résultats antérieurs avec CANO [3] (1970) en faisant remarquer que les taux respectifs de nicotine (32 µg/m<sup>3</sup>) et de CO (40 ppm) que nous avions mesurés sont dans un rapport très différent de celui d'ANDERSON et de DALHANU (1973) qui trouvèrent 0,577 mg/m<sup>3</sup> et 5 ppm et de HARKE (1972) qui obtenait 0,51 mg/m<sup>3</sup> et 64 ppm.

Il faut remarquer que, dans nos mesures en chambre étanche, nous avons également obtenu après fumage de 16 cigarettes pour 40 m<sup>3</sup> des concentrations de 0,517 mg/m<sup>3</sup> de nicotine et 50 ppm de CO et dans une pièce ordinaire fermée non ventilée 0,05 mg/m<sup>3</sup> et 5,6 ppm, ce qui confirme les résultats de HARKE et plus généralement les valeurs obtenues au cours d'essais de courte durée. Dans nos essais antérieurs avec CANO il s'agissait d'une exposition de très longue durée (plusieurs jours) et, conformément à ce qui a été dit de la décroissance spontanée de la concentration en nicotine il est normal que le taux moyen

2023380352

permanent soit resté assez bas tandis que le CO tendait à s'accumuler (et nécessitait même la mise en route périodique d'un absorbeur catalytique).

La faiblesse de l'imprégnation par voie aérienne du « fumeur passif » est confirmée par la détermination des quantités de nicotine excretées par voie urinaire. Dans notre travail antérieur avec Caxo (1970) nous avions montré que les sujets non-fumeurs séjournant 24 heures sur 24 dans une ambiance polluée par des fumeurs avec un taux atmosphérique moyen de 30 à 40 µg/m<sup>3</sup>, éliminaient environ 30 µg de nicotine dans les urines de 24 heures alors que les fumeurs en éliminaient environ 1 mg par jour. Ces résultats sont confirmés par le travail récent de RUSSO et FEVERAERT [19] (1975) qui trouvent chez le fumeur passif des taux urinaires environ 200 fois plus faibles que ceux des fumeurs.

Il semble raisonnablement prouvé par des enquêtes résumées par RIVIÈRE (1974) que les effets respiratoires de la fumée ne donnent pas lieu chez les non-fumeurs normaux à des différences significatives dans le taux des affections respiratoires. En ce qui concerne le problème des enfants, COURY [22] (1974) rapporte les résultats d'une enquête épidémiologique effectuée chez plus de deux mille écoliers et chez leurs parents. La fréquence de la toux chez les enfants paraît à première vue associée avec les habitudes tabagiques des parents. Une étroite corrélation existe entre les symptômes respiratoires des parents et des enfants. Mais lorsque les symptômes respiratoires des parents sont pris en compte, l'exposition des enfants à la fumée de cigarette produite par les parents a peu d'effets sur les symptômes des enfants. Ainsi, les associations entre les habitudes tabagiques des parents et les effets respiratoires sur les enfants rapportées par d'autres auteurs et interprétées comme liées à la pollution tabagique sont à rattacher en fait pour la majeure partie aux affections respiratoires des parents. Il est évident néanmoins qu'il faut déconseiller aux responsables de jeunes enfants de fumer dans les chambres où les enfants résident.

En revanche on ne peut nier que certains sujets hypersensibles soient incommodés par la fumée. On n'a pas pu mettre en évidence avec certitude des effets allergisants de la fumée (RIVIÈRE, 1974). Cependant il est indiscutable que chez une fraction importante de non-fumeurs l'exposition à la fumée se traduit au moins par des manifestations d'irritation oculaire et il est intéressant de rapporter les résultats de l'enquête menée en 1971 à l'occasion de voyages aériens par la Federal Aviation Administration et le N.I.O.S.H. aux U.S.A. [6]. Portant sur 3 296 passagers le questionnaire a fait apparaître, malgré la faible valeur de la pollution attestée par les dosages de CO, que 60 % des non-fumeurs et même 20 % des fumeurs étaient gênés par la fumée des autres passagers. Enfin, il faut souligner que de nombreux asthmatiques sont incommodés dans les ambiances ensuées, par un mécanisme qui nécessiterait une analyse plus approfondie ; il ne s'agit pas d'un mécanisme de nature allergique mais d'une augmentation de la sensibilité non spécifique des récepteurs bronchiques aux irritants de la fumée ou d'un phénomène psychosomatique.

## CONCLUSIONS

Les dosages que nous avons pratiqués dans des conditions réalistes montrent que dans l'atmosphère des lieux où l'on fume, les concentrations en monoxyde de carbone et composants non spécifiques de la fumée de tabac ne sont pas accrues dans une proportion suffisante pour qu'elles entraînent un risque toxicologique pour les non-fumeurs cohabituant avec les fumeurs.

En ce qui concerne la nicotine, sa concentration totale dans les atmosphères polluées par la fumée de tabac est souvent faible et le plus souvent inférieure à

2023380353

50 µg/m<sup>3</sup>. Si l'on suppose par exemple que le non-fumeur séjourne 10 heures par jour (travail + transport) dans une atmosphère en fumée à 50 µg/m<sup>3</sup> il inhalera 400 µg de nicotine dont il retient au plus 80 %, soit 320 µg. Si l'on rapproche cela de ce qu'inhale en moyenne un fumeur, soit environ 1 mg/cigarette, tout se passe comme si le non-fumeur avait fumé 40 % d'une seule cigarette pendant que le fumeur en fumait une vingtaine. Sur le plan strictement toxicologique on peut donc dire qu'il n'y a pas de risque tabagique pour les non-fumeurs.

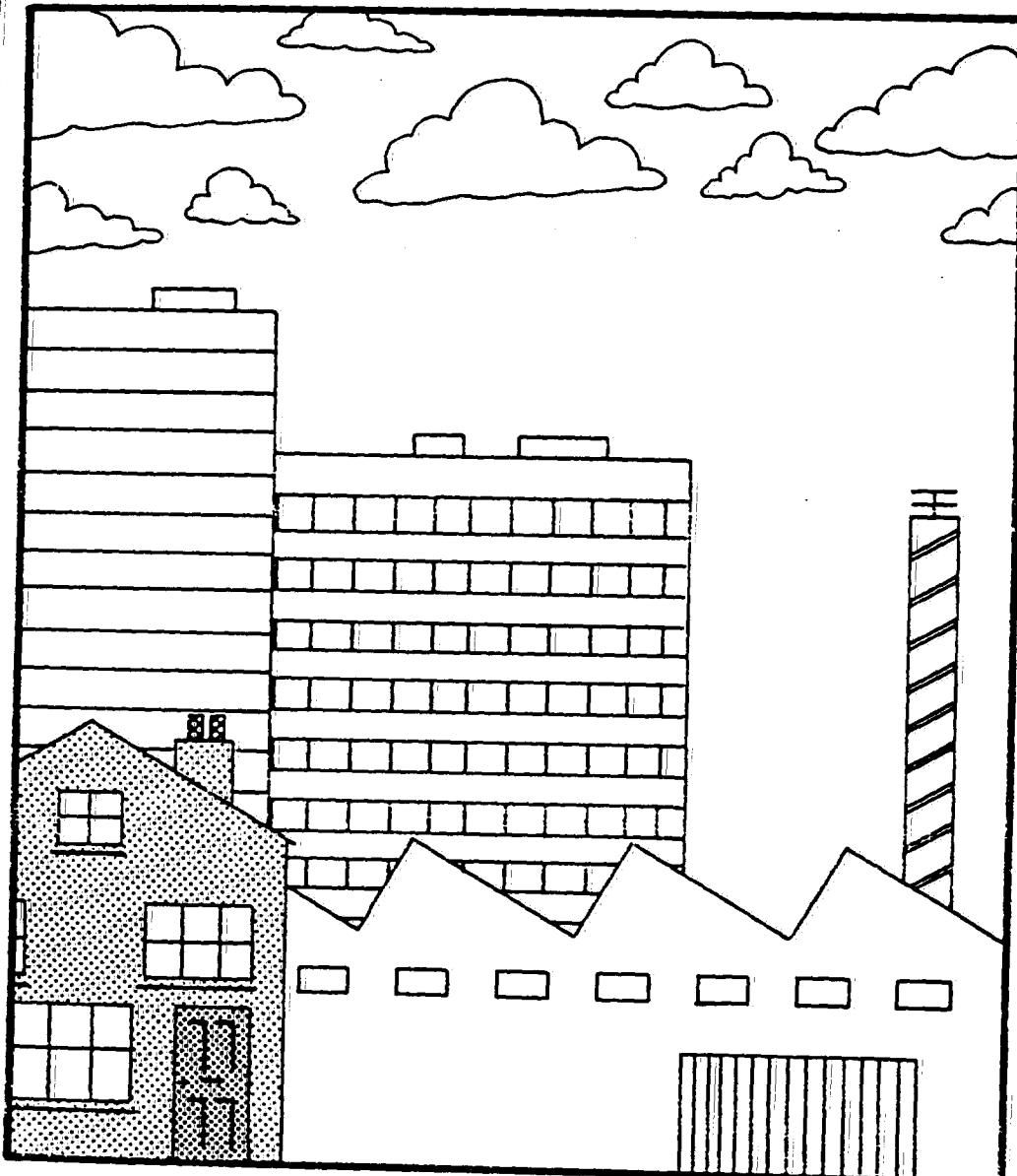
En raison notamment des incidences psychosomatiques, particulièrement nettes chez les asthmatiques, mais également possibles chez beaucoup de non-fumeurs, il est donc parfaitement déraisonnable d'inquiéter l'opinion publique par des affirmations sans fondement réel faisant du « fumeur passif » une victime dont la santé est menacée par la cigarette de son voisin. Il ne serait pas moins inadmissible que l'on ne tienne pas compte, et l'enquête de la F.A.A. l'a abondamment prouvé, de la gêne que représente pour une fraction très importante de la population, le plaisir que tirent leurs voisins de la satisfaction, en toute circonstance et quel que soit le lieu, de leur goût pour le tabac. On ne peut donc que souscrire aux initiatives tendant à renforcer ou à compléter les mesures de protection qui autrefois efficaces tendaient depuis quelques années à être négligées, notamment le respect des interdictions dans les transports en commun ou les compartiments réservés aux non-fumeurs.

#### BIBLIOGRAPHIE

- [1] BOURGUIN (M.), BADRE (R.) et DUMAS (C.). *Analusis*, 1975, 3, p. 34-38.
- [2] BRIDGE (D.P.) et CORN (M.). *Environmental Research*, 1972, 5, p. 192-209.
- [3] CANO (J.P.), CATALIN (J.), BADRE (R.), DUMAS (C.), VIALA (A.) et GUILLERM (R.). *Ann. pharm. fr.*, 1970, 28, p. 581-588.
- [4] COLE (P.V.) et BROWN (E.). *Lancet*, 1973, 1, p. 576-579.
- [5] DUKELOW (D.A.). *J.A.M.A.*, 1973, 223, p. 336-337.
- [6] Federal Aviation Administration (anonyme) (1971). *Health aspects of smoking in transport aircraft. Report U.S. Public Health Service Depart' of Health, Education and Welfare*.
- [7] GUILLERM (R.). *Les Cahiers Sandoz*, 1969, 15, p. 29-47.
- [8] GUILLERM (R.), BADRE (R.), HEE (J.) et MASUREL (G.). *Revue de Tuberculose et de Pneumologie*, 1972, 36, p. 187-208.
- [9] HARKE (H.P.). *Münch. Med. Wochr.*, 1970, 812, n° 51, p. 2328-2334.
- [10] HARKE (H.P.) et coll. *Int. Arch. Arbeitsmed.*, 1972, 29, p. 323-339.
- [11] HERNSHEN (H.) et EFFEKENBERGER (E.). *Arch. Hyg.*, 1957, 14, p. 383.
- [12] HESS (M.). *Literatur Eildiens*, Roche, 1959, 37, p. 49.
- [13] HINDS (W.C.) et FIRTS (M.W.). *New England J. Med.*, 1975, p. 844-845.
- [14] HORNING (E.C.) et coll. *Life Sciences*, 1973, 13, p. 1331-1346.
- [15] HUBER (G.). *New England Journal Med.*, 1975, p. 858-859.
- [16] KEIT (C.), DERRICK (J.). *J. Coll. Sc.*, 1960, 15, 340-356.
- [17] NAUMLINN (R.J.). *Sciences*, 1973, 182, n° 4110, p. 334-336.
- [18] RYLANDER (R.). *Scandinavian Journal of Resp. Diseases*, 1974, Supplm. n° 9).
- [19] RUSSEL (M.A.) et FEYERABEND (C.). *Lancet*, 1975, 1, p. 79-81.
- [20] SCASSELATI et coll. *Riv. Ital. Igiene*, 1958, 28, p. 43-55.
- [21] TAYLOR (G.). *Scand. J. Resp. Dis.*, 1974, Suppl. n° 91, p. 50-55.
- [22] COLLET (J.R.T.). *Brit. med. Journal*, 1974, 2, p. 201-203.

2023380354

# INDOOR AND AMBIENT AIR QUALITY



2023380355

Edited by R. Perry  
and P.W. Kirk



# **INDOOR AND AMBIENT AIR QUALITY**

**Sold and distributed by Publications Division,  
Selper Ltd., 79 Rusthall Avenue,  
Chiswick, London W4 1BN**

**ISBN 0 948411 04 X**



**Copyright held by  
©Selper Ltd., 1988**

**Published by  
Publications Division, Selper Ltd., London  
Printed by Printext Ltd., London**

**2023380356**

# DEVELOPMENT AND APPLICATION OF A THERMAL DESORPTION-BASED METHOD FOR THE DETERMINATION OF NICOTINE IN INDOOR ENVIRONMENTS

R.A. Jenkins, C.V. Thompson & C.E. Higgins

Organic Chemistry Section, Analytical Chemistry Division, Oak Ridge National Laboratory, Oak Ridge, Tennessee 37831-6124, USA

## ABSTRACT

A personal monitoring system for the determination of exposure to nicotine has been developed. The system consists of a sampling cartridge packed with 200 mg of Tenax GC<sup>R</sup> and a small, constant flow, personal sampling pump. After sampling, the cartridges are analyzed by triethylamine-assisted thermal desorption gas chromatography with nitrogen-selective detection. Collection and desorption efficiencies for the cartridges have been determined. The system has been applied in a variety of work sites, and in 36 restaurants, where measured concentrations of nicotine ranged from 0.5 to 37.2  $\mu\text{gm}^{-3}$ .

## INTRODUCTION

Environmental tobacco smoke (ETS), comprised of diluted and aged side-stream smoke mixed with main-stream smoke exhaled by the smoker, represents a potentially significant contribution to indoor air pollution. Concentrations of ETS respirable suspended particulates (RSP) have been reported to range from 0 to 700  $\mu\text{gm}^{-3}$  in indoor environments (1). A number of procedures have been applied for estimating ETS concentrations based on the measurement of concentrations of particular ETS constituents, such as CO (2-5), oxides of nitrogen ( $\text{NO}_x$ ) (3-5), and particulate matter (4-9). However, these constituents of tobacco smoke are also the products of other combustion processes, an aspect which limits their utility as markers for estimating ETS levels, especially in complex atmospheres such as those which exist in indoor environments. In contrast, nicotine's uniqueness makes it a likely marker for ETS.

Several methods have been developed for determining nicotine concentrations at fixed sampling locations in industrial settings (10,11). However, these are of limited utility in more conventional environments, due to their relatively high limits of detection (LOD). Other methods reported in the literature for non-industrial applications include the use of cold Petri dishes (12), untreated glass fiber filters (4) or diffusion denuder tubes (13) for collection of ambient nicotine.

The development and testing of a number of personal monitoring systems which measure individual exposures to ETS as determined by ambient nicotine concentrations have been reported recently in the literature. Solvent desorption-based systems include personal sampling pumps coupled with commercially available XAD-4 cartridges (14, 15, 16) and  $\text{NaHSO}_4$ -treated, Teflon-coated glass fiber filters (17), and a passive sampling system utilizing the treated filters (18). The limitation of using solvent

N  
2023380357

extraction of samples is that only a small fraction of the analyte is actually analyzed. This necessarily raises the theoretical LOD for such methods relative to those such as thermal desorption, that use all of the acquired sample. Two thermal desorption-based personal monitoring systems for nicotine have been reported, one by Proctor (19) that employs an unspecified adsorbent and analysis system and another by Muramatsu et al. (20, 21), that utilizes an ammonia purge of the sample cartridge during desorption into a gas chromatograph (gc). This method requires modification of the gc and desorption system by placing an ammonia bubbler in-line with the carrier gas.

This paper discusses the development and application of a thermal desorption-based personal monitoring system for nicotine using Tenax GC as the adsorption material. This system is similar in concept to the system developed by Muramatsu; its difference - and advantage - is that it lacks the mechanical complexities of the ammonia purge during desorption and relies on a more commonly available trapping medium.

#### EXPERIMENTAL

**Personal Monitoring Systems** - The air sampling cartridges developed consisted of 16 cm sections of 4 in. O.D. borosilicate glass tubing which was treated with NH<sub>4</sub>OH and packed with approximately 200 mg of Tenax GC, 35-60 mesh. Before use, cartridges were conditioned at 250°C, in a stream of N<sub>2</sub> flowing at 40 mlmin<sup>-1</sup> for at least two hours. The cartridges could be reused after washing (with 2-3 mL of methanol) and subsequent thermal reconditioning.

Alpha-2 personal sampling pumps, available from DuPont (Kemett Square, PA), were used for sample collection in most experiments and were chosen for their light weight (410 g) and low-noise level during operation. For experiments performed in the chambers and offices, pumps were connected to Tenax cartridges with a section of flexible tubing, and air from the area sampled was drawn through the cartridge. For sampling conducted in restaurants, the pumps were worn under jackets and the sampling tubes placed unobtrusively within 25 cm of the mouth of the individual doing the sampling. All samples were collected for at least one hour with the pump operating at a flow rate of 170 mlmin<sup>-1</sup>. The cartridge was refrigerated at 3°C until analysis.

**Analysis Procedure** - Nicotine standards were prepared by diluting redistilled nicotine (98%) in ethyl acetate which contained 0.01% triethylamine (TEA) (19). Internal standards employing quinoline were prepared by diluting quinoline in a solution of ethyl acetate/5% TEA. Analyses were performed with a gas chromatograph equipped with a nitrogen/phosphorous detector (gc/NPD) and a 2 m x 2 mm i.d. glass column packed with 10% Carbowax 20M/2% KOH on 80-100 mesh Chromosorb W-AW. The injector and detector were set at 250°C and the column oven was programmed from 70°C to 175°C at 46°C/min., following an initial 8 minute hold. Typically, a calibration curve was generated from the desorption of 9 duplicate sets of Tenax traps loaded with amounts of nicotine ranging from 1.5 to 700 ng and with 250 ng of quinoline internal standard.

**Atmospheres Sampled** - The initial experimental atmospheres for the development of the Tenax method were generated in two stainless steel chambers with volumes of 0.4 and 1.4 m<sup>3</sup>. Side-stream smoke from a 2R1 Kentucky Reference cigarette (procured from the University of Kentucky)

2023380358

Tobacco and Health Research Institute, Lexington, KY) smoldering in a laminar flow smoke generator (22), was pulled into the smaller chamber at a rate of 30 Lmin<sup>-1</sup> and diluted with an air flow of 250 to 1000 Lmin<sup>-1</sup>, the exact rate depending on the concentration of ETS needed. Concentrations of particulate matter in the chambers were monitored with a TSI-5000 piezoelectric balance (TSI, St. Paul, MN) and an RAS-1 light scattering sensor (CCA Instruments, Bedford, MA), which was modified in our laboratory to enhance its sensitivity. The nicotine and PM concentrations utilized for these experiments are much higher than what would be typically observed in real life situations and were used only to determine the potential utility and the upper analytical limits of the method. After development experiments involving the chamber were concluded, other experiments were conducted in an un-occupied office at ORNL. Diluted sidestream smoke designed to simulate ETS was produced by smoking 1R4F Kentucky Reference cigarettes at one 35 mL puff per minute on an ADL-II smoking machine (from Arthur D. Little Co., Cambridge, MA). Mainstream smoke was collected in sealed Tedlar bags. Again, PM levels were monitored with a TSI-5000 piezoelectric balance.

Additional laboratory evaluations of the method's performance were conducted in an 18 m<sup>3</sup> environmental chamber (23) used for ETS studies and located at the R. J. Reynolds Tobacco Company's facilities in Winston-Salem, NC. PM concentrations in the chamber were monitored with a TSI-5000 piezoelectric balance. Initial field evaluations were conducted in work areas, offices, common areas, and dining areas at Oak Ridge National Laboratory. Field sampling was conducted in restaurants selected at random from those in the Knoxville, TN Standard Metropolitan Statistical Area (SMSA) (Knox, Blount, and Anderson counties).

#### RESULTS AND DISCUSSIONS

Results of initial experiments with Tenax cartridges showed evidence of incomplete desorption of nicotine, with up to 10% of the nicotine remaining on the cartridge. In order to enhance nicotine desorption, an internal standard solution was prepared which included 5% TEA. Internal standard spikes thus contained about 200 µg of TEA, which, as a stronger base, was thought to displace nicotine from acidic sites within the sampling cartridge or analysis train.

Experiments conducted in the 0.4 and 1.4 m<sup>3</sup> chambers were performed to determine the functional capabilities of the method and the nicotine collection efficiency. Typically, nicotine and PM concentrations ranged from lows of 34 and 74 µgm<sup>-3</sup>, respectively, to highs of 302 and 757 µgm<sup>-3</sup>, respectively. The ratios of nicotine to particulate matter in these experiments were substantially higher than what has been reported in typical indoor environments (24). However, this discrepancy was judged of little consequence since investigation of nicotine levels was the sole focus of the study.

Experiments to determine sample volumes at which nicotine breakthrough became significant were conducted by placing two Tenax cartridges in series and sampling from simulated high concentration ETS environments in the chambers. Results indicated not more than 1% breakthrough for sample volumes ranging from 20 to 45 L and nicotine concentrations ranging from 70 to 250 µgm<sup>-3</sup>. At lower sample volumes, breakthrough percentages are expected to be correspondingly lower.

In Table 1 are listed the results from sampling of highly diluted sidestream smoke in an un-occupied office. This range of nicotine and PM

2023380359

levels more closely approximated that which would be expected from sampling in public places. The correlation between nicotine and particulate levels was particularly high in this experiment, with the correlation coefficient of 0.976 for a first order regression analysis of these two parameters.

TABLE 1: CONCENTRATIONS OF NICOTINE AND PARTICULATE MATTER IN HIGHLY DILUTED SIDESTREAM SMOKE MEASURED IN AN UN-OCCUPIED OFFICE

TRIAL NO.	PARTICULATE MATTER ( $\mu\text{gm}^{-3}$ )	NICOTINE CONCENTRATION ( $\mu\text{gm}^{-3}$ )
1	14.6	1.8
2	23.2	1.9
3	28.2	2.4
4	15.2	3.7
5	58.6	4.8
6	59.6	8.2
7	113.0	21.7
8	115.4	27.3
9	257.0	48.3
10	248.8	49.0

The detection limit of the method was determined by sampling, in triplicate, a very dilute environment of ETS generated in the office [air exchange rate = 2.4 ACH (air exchanges per hour)] with one puff and one minute of smoking from a 1R4F Kentucky reference cigarette. Following a one hour sampling period, analysis of the Tenax cartridges showed an average of  $3.0 \pm 0.3$  ng per cartridge, corresponding to  $0.3 \mu\text{gm}^{-3}$  nicotine, with a relative standard deviation (RSD) of 10%. A second experiment conducted at a slightly lower ETS concentration gave an average nicotine loading per cartridge of  $2.3 \pm 0.6$  ng corresponding to a concentration of  $0.2 \mu\text{gm}^{-3}$  and an RSD of 26%. This level of variation was arbitrarily considered to be unacceptable for the method, so the detection limit was defined as  $0.3 \mu\text{gm}^{-3}$  for a 10 L sample. In practice, the detection limit may be as low as  $0.08 \mu\text{gm}^{-3}$  if the sample volume were increased to 40 L.

In Table 2 are listed the results from a sampling study conducted in an  $18 \text{ m}^3$  chamber at R. J. Reynolds. An evaluation of the method for ambient nicotine developed by Ogden et al (14) was being conducted simultaneously. The object of the study was to examine relative method performance at low ETS levels. In general, the data indicated that agreement between the two methods was excellent.

Listed in Table 3 are the results from samples collected at Oak Ridge National Laboratory facilities which amounted to an in-house field test of the method. The arithmetic mean and standard deviation of nicotine concentrations for all sample sites is  $10.5 \pm 17.2 \mu\text{gm}^{-3}$ . However, the relatively high concentrations measured in the first common area ( $36.5 \pm 18.1 \mu\text{gm}^{-3}$ ) influence the average disproportionately. As can be seen in Figure 1, the data appear to be distributed in a log normal pattern; thus the geometric mean of  $3.2 \mu\text{gm}^{-3}$  may be more appropriate for this data set.

2023380360

TABLE 2: RESULTS FROM DETERMINATIONS OF NICOTINE  
BY TENAX AND XAD-4 METHODS  
MEAN NICOTINE CONCENTRATION. ( $\mu\text{gm}^{-3}$ )

RUN No.	Particulate Matter Concentration ( $\mu\text{gm}^{-3}$ )	TENAX <sup>a</sup>	ORNL <sup>b</sup> XAD-4	RJR (A) <sup>c</sup> XAD-4	RJR (B) <sup>c</sup> XAD-4
1	55	2.5	-	-	-
2	14	1.8	-	-	-
3	103	9.0	-	-	-
4	62	4.1	4.1	4.3	4.3
5	16	2.1	2.1	2.2	2.3
6	128	5.5	5.5	5.9	5.7

(a) N = 3 determinations. (b) Analyzed by the authors using the method developed at R. J. Reynolds Tobacco Co. N = 2 determinations. (c) Analyzed at R. J. Reynolds Tobacco Co. (30). N = 2 determinations. RJR (A) analyzed using 0.53 mm i.d. capillary column with direct injection. RJR (B) analyzed using 0.32 mm i.d. capillary column with split injection.

TABLE 3  
NICOTINE CONCENTRATIONS MEASURED AT SELECTED LOCATIONS  
WITHIN OAK RIDGE NATIONAL LABORATORY

LOCATION	AMBIENT NICOTINE LEVEL ( $\mu\text{gm}^{-3}$ )	LOCATION	AMBIENT NICOTINE LEVEL ( $\mu\text{gm}^{-3}$ )
Offices	4.2 $\pm$ 0.1 <sup>a</sup>	Common Area	30.0 $\pm$ 0.9
	4.0 $\pm$ 3.5 <sup>a</sup>		60.3 $\pm$ 2.1
	4.5 $\pm$ 0.5 <sup>a</sup>		53.1 $\pm$ 2.8 <sup>a</sup>
	6.7 $\pm$ 0.9		23.2 $\pm$ 2.9
	0.7 $\pm$ 1.0		39.7 $\pm$ 0.1
	1.1 $\pm$ 1.5		12.6 $\pm$ 1.2
	0.6 $\pm$ 0.8		3.8 $\pm$ 0.1
	0.6 $\pm$ 0.9		2.2 $\pm$ 0.9
	0.3 $\pm$ 0.4		1.0 $\pm$ 0.4
Dining Area	4.4 $\pm$ 0.8 2.3 <sup>b</sup>		0.9 $\pm$ 1.3 1.7 $\pm$ 0.8 0.8 $\pm$ 1.1
Work Area	2.0 $\pm$ 0.3		

(a) N = 3 determinations. (b) N = 1. All others, N = 2 determinations.

2023380361

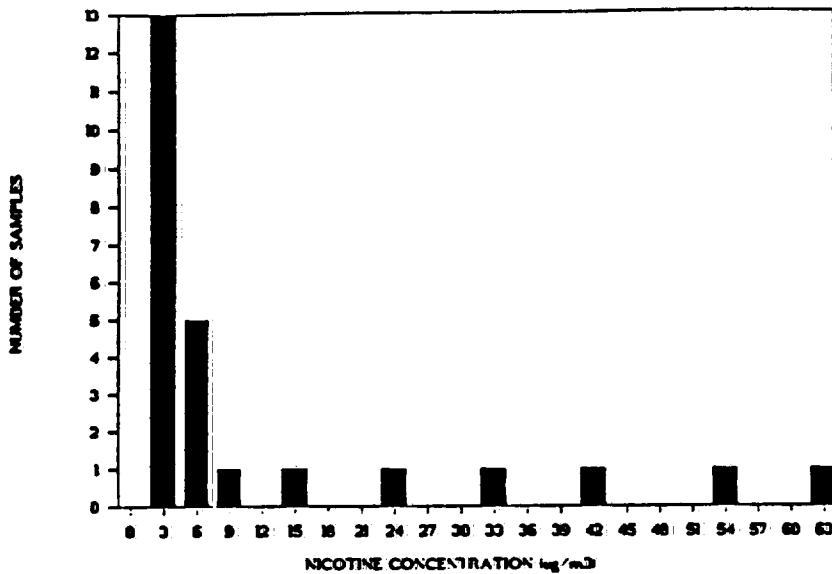


Figure 1. Distribution of nicotine levels for work sites at ORNL. Note that nicotine concentrations listed are the maxima for the individual cells.

Results from the field determinations of nicotine concentrations in the randomly selected restaurants are shown in Table 4. Samples 28 and 29 were acquired simultaneously from the same restaurant, with an RSD for these two samples of 1%, suggesting good reproducibility for the method. Nicotine concentrations found in the restaurants ranged from 0.5 to 37.2  $\mu\text{gm}^{-3}$  with an arithmetic mean of  $5.6 \pm 6.4 \mu\text{gm}^{-3}$ . As in the treatment of the data from the in-house sampling and analysis, a plot of the distribution of the concentration data indicates that it fits a log normal rather than Gaussian pattern (Fig. 2). The geometric mean was computed to be  $3.5 \mu\text{gm}^{-3}$ , while the 95% confidence boundaries on the median of the distribution were 2.5 and 4.8  $\mu\text{gm}^{-3}$ . By way of comparison, in Table 5 are listed selected results from studies performed by other investigators in a number of different public environments. In general, our results are comparable to those reported by these investigators.

Major factors likely to affect nicotine concentrations in a restaurant are the number of cigarettes smoked, the volume of the room, the proximity of smokers to the sample location, and the air exchange rate (data relative to this last factor were not acquired). The Pearson's correlation coefficients (*r*) for first order regressions of nicotine concentration to number of cigarettes, number of smokers, restaurant volume, and distance to the closest smoker are 0.669, 0.783, 0.049, and -0.155, respectively. The significance

2023380362

TABLE 4: ETS NICOTINE LEVELS IN RESTAURANTS

SAMPLE NUMBER	# SMOKERS OBSERVED	# CIGARETTES	# CIGARS OR PIPES	ESTIMATED RESTAURANT VOLUME (m <sup>3</sup> )	CLOSEST SMOKER (ft.)	NICOTINE CONCENTRATION (μg m <sup>-3</sup> )
34	0	0		179	-	0.5
16	0	0		595	-	0.5
24	1	1		198	6	0.7
21	1	1		41	9	0.8
10	0	0		638	-	1.1
20	7	9		227	5	1.4
31	2	2		272	12	1.5
14	1	2		283	20	1.5
25	1	1		136	8	1.6
18	2	2		1204	15	2.3
19	0	0		453	-	2.3
27	2	2		317	10	2.4
29	6	8		1700	10	2.4
9	8	9		510	5	2.5
2	7	8		113	8	3.3
26	6	7		213	2	3.3
22	4	3	1	170	3	3.5
11	12	14		204	5	4.1
12	9	9		1785	7	4.2
15	4	4		198	5	4.3
35	10	11		623	4	4.5
32	5	7		397	5	4.8
33	6	10		1063	14	4.8
23	5	8	1	238	10	4.9
7	8	11		2380	5	5.6
1	15	18	1	744	2	5.7
6	6	8		340	2	5.9
4	4	3		179	7	7.3
5	4	3		179	7	7.4
17	19	35	1	680	4	7.8
28	10	15		204	5	8.0
13	9	10		177	6	9.3
30	19	25		793	4	12.1
3	33	43		1666	5	12.6
8	14	18		680	5	13.5
36	-	30		272	8	37.2

2023380363

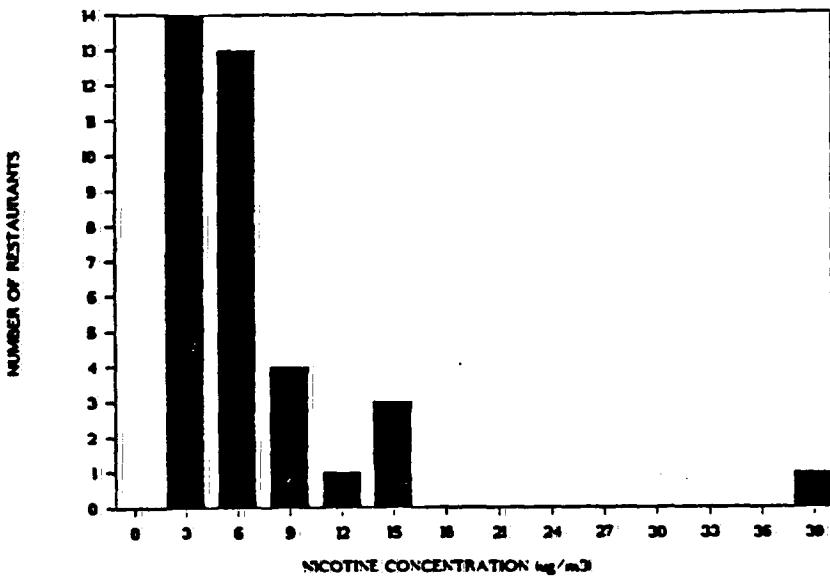


Figure 2. Distribution of nicotine levels in selected Knoxville, Tennessee area restaurants. Note that nicotine concentrations listed are the maxima for the individual cells.

TABLE 5: PREVIOUSLY REPORTED ETS NICOTINE LEVELS IN PUBLIC PLACES

LOCATION	RANGE OF OBSERVED NICOTINE LEVELS ( $\mu\text{g}/\text{m}^3$ )	MEAN (IF REPORTED) ( $\mu\text{g}/\text{m}^3$ )	INVESTIGATORS	REFERENCE
Restaurants	7.1 - 27.8	14.8	Muramatsu, et al	20
Restaurants	-	5.2	Hinds and First	25
Restaurants	0 - 24	5	Oldaker, et al	16
Offices	3 - 48	-	Hammod, et al	17
Offices	9 - 32	-	Muramatsu, et al	20
Offices	6 - 20	-	Muramatsu, et al	21
Offices	0 - 70	5	Oldaker, et al	?
Common Areas	2 - 36	-	Muramatsu, et al	20
Common Areas	1 - 3	-	Hinds and First	25
Aircraft Smoking Sections	6 - 29	15	Muramatsu, et al	21
Aircraft Smoking Sections	0 - 112	9	Oldaker & Conrad	15
Aircraft Non-Smoking Sections	0 - 40	6	Oldaker & Conrad	15

2023380364

levels (*p*) for the regressions are 0.0001, 0.0001, 0.776, and 0.396, respectively. A correlation is thus indicated between nicotine concentration and both the number of cigarettes and number of smokers. If sample 36 is eliminated from the regression analysis for nearest smoker, then *p* = 0.03, indicating a correlation between nicotine concentration and this parameter.

#### CONCLUSIONS

A method for the determination of personal exposure to concentrations of nicotine in indoor environments has been developed. The method has a low detection limit and can be used unobtrusively. The procedure has been applied to the determination of nicotine concentrations in a number of restaurants and the results seem comparable to those obtained by other researchers utilizing different methods for sampling in restaurants and other public places.

#### ACKNOWLEDGEMENTS

Support for this work was provided by the Council for Tobacco Research, Inc., under Interagency Agreement DOE No. ERD-85-471, CTR Project No. 132 with Martin Marietta Energy Systems, Inc., under Contract DE-AC05-84OR21400 with the U.S. Department of Energy. We thank J. H. Moneyham for his assistance in generation of simulated ETS atmospheres in the stainless steel chambers and in sample acquisition. We also thank C. D. Varnadore for his assistance in sample acquisition.

#### LITERATURE CITED

- (1) J. D. Brain and B. E. Barry, Indoor Air and Human Health. Proceedings of the Seventh Life Sciences Symposium, R. B. Gammie and S. V. Kaye, Eds., Lewis Publishers, Inc., Chelsea, MI, pp 215-225 (1985).
- (2) R. Badre, R. Guillerm, M. Abran, M. Bourdin and C. Dumas, Ann. Pharm. Fr., 36, 443-452 (1978).
- (3) T. Fischer, A. Weber, and E. Grandjean, Int. Arch. Occup. Environ. Health, 41, 267-280 (1978).
- (4) A. Weber and T. Fischer, Int. Arch. Occup. Environ. Health, 47, 209-221 (1980).
- (5) C. Hugod, L. H. Hawkins, and P. Astrup, Int. Arch. Occup. Environ. Health, 42, 21-29 (1980).
- (6) J. L. Repace and A. H. Lowrey, Science (Washington, DC), 208, 464-472 (1980).
- (7) J. D. Spangler, R. D. Treitman, T. D. Testason, D. T. Mage and M. L. Soczek, Environ. Sci. Technol., 19, 700-707 (1985).
- (8) J. L. Repace and A. H. Lowrey, Environ. Int., 11, 3-22 (1985).
- (9) F. R. Quant, P. A. Nelson, and G. J. Sam, Environ. Int., 8, 223-227 (1982).

2023380365

- (10) Manual of Analytical Methods; National Institute for Occupational Safety and Health: Cincinnati, OH, Part II, Vol. 3, Method No. S293 (1977).
- (11) U. E. Crouse, L. F. Johnson, and R. S. Marmor, Beitr. Tabakforsch., 10, 111-113 (1980).
- (12) D. C. Williams, J. R. Whitaker, and W. G. Jennings, Environ. Health Perspect., 60, 405-410 (1985).
- (13) D. J. Estough, C. Benner, R. L. Mooney, D. Bartholomew, D. S. Steiner, L. D. Hansen, J. D. Lamb, E. A. Lewis, and N. L. Estough; Presented at 79th Annual Meeting of the Air Pollution Control Association, Minneapolis, MN (June, 1986).
- (14) M. W. Ogden, L. W. Eudy, D. L. Heavner, F. W. Conrad and C. R. Green; Presented at the 40th Tobacco Chemists' Research Conference, Knoxville, TN (Oct., 1986).
- (15) G. B. Oldaker, and F. C. Conrad,Jr., Environ. Sci. Technol., 21, 994-999 (1987).
- (16) G. B. Oldaker, P. F. Perfetti, F. C. Conrad, Jr., J. M. Conner and R. L. McBride; Presented at the International Conference on Indoor Air Quality, Tokyo, Japan (Nov., 1987).
- (17) S. K. Hammond, B. P. Leaderer, A. C. Roche and M. Schenker, Atmos. Environ., 21, 457-461 (1987).
- (18) S. K. Hammond and B. P. Leaderer, Environ. Sci. Technol., 21, 497-500 (1987).
- (19) C. Proctor in Toxicol. Lett., 35, 131-134 (1987).
- (20) M. Muramatsu, S. Umemura, T. Okada, and H. Tomita, Envir. Res., 35, 218-227 (1984).
- (21) M. Muramatsu, S. Umemura, J. Fukui, T. Arai and S. Kira, Int. Arch. Occup. Environ. Health., 59, 545-550 (1987).
- (22) J. H. Moneyhun, R. A. Jenkins and M. R. Guerin; Presented at the 40th Tobacco Chemists' Research Conference, Knoxville, TN (Oct., 1986).
- (23) D. L. Heavner, F. A. Thome, L. W. Eudy and C. A. Green; Presented at the 39th Tobacco Chemists' Research Conference, Montreal, Quebec, Canada (Oct., 1985).
- (24) G. B. Oldaker, J. M. Conner, F. W. Conrad, Jr., B. C. McConnell, M. W. Ogden, P. F. Perfetti and M. W. Stancill; Presented at the 41st Tobacco Chemists' Research Conference, Greensboro, NC (Oct., 1987).
- (25) W. C. Hinds, and M. W. First, N. Eng. J. Med., 292, 844-845 (1975).

2023380366

## Estimation of Personal Exposure to Tobacco Smoke with a Newly Developed Nicotine Personal Monitor

MOTOHIKO MURAMATSU, SETSUOKO UMEMURA, TAKASHI OKADA, AND  
HIDEO TOMITA

Central Research Institute, The Japan Tobacco & Salt Public Corporation, 6-2 Umegasaki,  
Midori-ku, Yokohama, Kanagawa 227, Japan

Received August 16, 1983

To evaluate the actual level of exposure of nonsmokers to tobacco smoke in their living environments, a convenient personal monitor of nicotine specific for tobacco smoke has been developed. The nicotine personal monitor consists of a sampler tube containing 450 mg of Unipon-S coated with silicon OV-17 and a portable sampling pump with a mechanical counter for obtaining total sampling volume. Using the personal monitor attached to a nonsmoker, ambient nicotine was collected in the sampler tube by drawing environmental air at a constant flow rate for a maximum period of 8 hr. The collected nicotine was desorbed by heating and directly transferred onto a GC column with a carrier gas. The amounts of nicotine inhaled by passive smoking in various living environments were estimated to be in the range of 0.9-40 µg/hr. These levels are equivalent to those from the active smoking of about 0.091±0.044 ordinary cigarettes in 1 hr.

### INTRODUCTION

In studying the influence of passive smoking, it is fundamentally important to determine the actual level of personal exposure of nonsmokers to tobacco smoke in their usual living environments.

In previous studies, the level of passive smoking has been evaluated on the basis of concentrations of (a) indoor air pollutants such as particulate matter (9, 20), CO (2, 5, 9, 19, 20), and nicotine (2, 8, 9, 20) measured at a fixed monitoring station, and (b) some constituents in biological samples such as COHb (1, 6, 9, 14, 15), blood and urinary nicotine (4, 6, 16), and urinary hydroxyproline, HOP (12). However, these methods are not accurate enough to evaluate quantitatively the actual level of personal exposure to tobacco smoke, because (a) concentrations of particulate matter, CO, COHb, and HOP are affected by other air pollutants as well as tobacco smoke; (b) inhaled nicotine is reduced by half in a short time in the human body (13), though nicotine is specific for tobacco smoke; and (c) fixed monitoring does not entirely represent the actual level of air pollutants that people encounter in their daily lives.

In the present work, a pocketable personal nicotine monitor specific for tobacco smoke has been developed and applied to the estimation of the actual level of passive smoking in the usual living environments. The results showed that the amounts of tobacco smoke inhaled by nonsmokers were very small compared with those of active smokers.

0013-9351/84 \$3.00  
Copyright © 1984 by Academic Press, Inc.  
All rights of reproduction in any form reserved.

2023380367

Persi  
minatic  
of pers  
measur  
person  
The  
pling p  
volume  
a sampl  
Unipo  
sorbent  
with th  
gas thi  
Usin  
collect  
a flow  
the co  
of 7-n  
stand:  
280°C  
gas ci  
carrie  
tube;  
GC co  
column  
trap a  
the fi  
three  
moni  
for th  
port-  
A

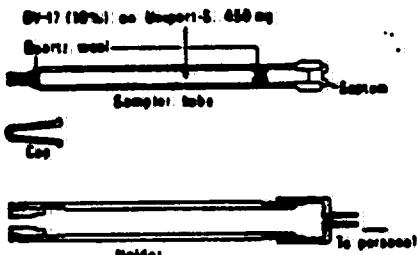


FIG. 1. Details of sampler tube.

#### MATERIALS AND METHODS

*Personal nicotine monitoring system.* The methods so far used for the determination of ambient nicotine (2, 3, 8, 18, 20, 21) are inadequate for the evaluation of personal exposure to nicotine from the viewpoint of sensitivity and facility of measurement and portability of sampling device. The newly developed nicotine personal monitor has overcome these defects as described below.

The personal monitor consists of a sampler tube and a small, light-weight sampling pump (about 340 g) with a mechanical counter for obtaining total sampling volume. The personal monitor can be carried conveniently by a person throughout a sampling period. The details of the sampler tube are shown in Fig. 1. The sampler tube, made of Pyrex glass (12 cm long, 6 mm i.d.), contains 450 mg of Uniport-S (60-80 mesh) coated with 10 wt% of silicon OV-17 as a nicotine absorbent. This absorbent was selected after several trials. The sampler tube packed with the absorbent was aged at 310°C for more than two days by passing nitrogen gas through the tube at 40 ml/min.

Using the personal monitor attached to a nonsmoker, ambient nicotine was collected on the sampler tube by drawing environmental air through the tube at a flow rate of 40 ml/min for a period of 1 to 8 hr unless otherwise specified. After the collection of ambient nicotine, 5 µl of n-propanol solution containing 400 ng of 7-methylquinoline (7-MQ) was injected into the sampler tube as an internal standard. Then, the sampler tube was placed in a cylindrical furnace heated at 280°C and one end of the tube was quickly connected to the injection port of a gas chromatograph (GC) via a needle with a side hole and the other end to the carrier gas bypass shown in Fig. 2. Passing the carrier gas through the sampler tube, collected nicotine and 7-MQ were desorbed and directly transferred onto a GC column. Thermal desorption was allowed to continue for 8 min, while the GC column temperature was held at 70°C during the desorption period in order to trap and concentrate both nicotine and 7-MQ on the top of the column. During the first 3 min of thermal desorption a small amount of ammonia vapor was added three times into the carrier gas by bubbling the carrier gas through 35 wt% ammonia water for 6 sec. once per min. The addition of ammonia vapor is effective for thermal desorption of nicotine and 7-MQ against the acidic property of Uniport-S.

A Teflon sealing tape used to bring the needle and the sampler tube into tight

2023380368

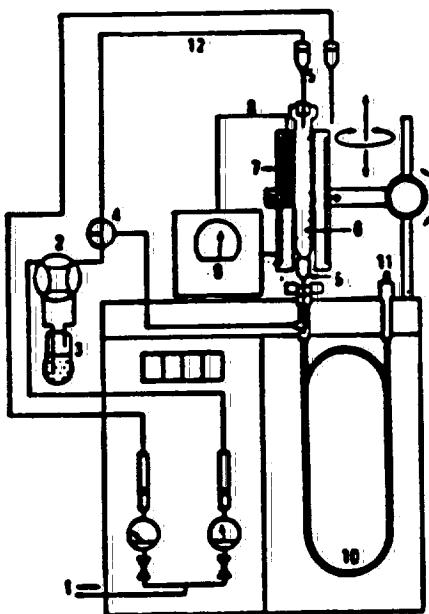


FIG. 2. Schematic diagram of apparatus for measurement of ambient nicotine. (1) N<sub>2</sub> inlet; (2) 4-way cock; (3) reservoir for NH<sub>4</sub>OH; (4) 3-way cock; (5) needle; (6) sampler tube; (7) beaker; (8) thermocouple; (9) temperature regulator; (10) column; (11) FTD; (12) bypass.

contact was treated at 320°C before use until a ghost peak attributable to the tape disappeared on the gas chromatogram.

**Gas chromatography.** A Hitachi 663-30 GC equipped with a nitrogen-sensitive detector was employed under the following conditions.

Column: 2 m × 3 mm i.d. glass column packed with Chromosorb W (AW-DMCS, 30–60 mesh) coated with 10 wt% of PEG-20M and 2 wt% of KOH.

Column temperature: initially maintained at 70°C for 8 min and then programmed to 185°C at 46°C/min and maintained at 185°C until completion of elution.

Detector and injection temperature: 300°C.

Carrier gas: nitrogen: 40 ml/min.

Additional gas: nitrogen: 7.5 ml/min; air: 75 ml/min; hydrogen: 1.5 ml/min.

Bead current: 1.55–1.60 A.

The time required for GC analysis per test sample is about 20 min, including the thermal desorption period. Peak areas on the gas chromatogram were measured with a digital integrator (Takedaiken Inc. Co. Ltd., Model 2213).

**Calibration curve.** The calibration curve of nicotine was prepared by use of a series of n-propanol solutions containing widely different amounts of nicotine (0–100 µg/ml) and a constant amount of 7-MQ (80 µg/ml). After the injection of 5 µl of each solution into the sampler tube, nicotine and 7-MQ were desorbed and directly transferred onto the GC column for analysis as described above. A plot of the peak area ratio of nicotine/7-MQ to the amount of nicotine gave a good linear line over a wide range of nicotine from 0 to 500 ng.

2023380369

TABLE I  
COLLECTION EFFICIENCY OF AMBIENT NICOTINE ON SAMPLER TUBE

Sampling rate (ml/min)	Run	Amount of nicotine collected (ng)		Collection efficiency [(A - B)/A, %]
		A (first)	B (second)	
40	1	147.8	1.8	98.7
	2	253.9	3.9	98.7
	3	139.9	1.5	98.9
	4	153.9	6.0	96.1
100	5	310.2	1.4	99.5
	6	492.0	6.7	98.6
	7	298.5	1.6	99.4
	8	194.6	3.8	98.0

*Personal sampling pump.* A MDA 808 (MDA Scientific Inc.) personal sampling pump was used. The sampling volume per stroke of the pump was calibrated for the individual sampler tubes under actual operating conditions, using a gas flow meter (Shinagawa Keisoku Seisakusho Inc., Model WK-0.5).

## RESULTS AND DISCUSSION

### Collection Efficiency of Ambient Nicotine

The collection efficiency of ambient nicotine on the sampler tube was examined by drawing environmental air at 40 or 100 ml/min for 8 hr through the two sampler tubes connected in series. Table I shows the amounts of nicotine collected on the first tube, A, and the second tube, B, as well as the collection efficiency of ambient nicotine per sampler tube. The collection efficiency was calculated from equation  $(A - B)/A$  by assuming that the amounts of nicotine collected on the individual sampler tubes connected indefinitely would be represented by a geometric progression.

More than 98.5% of nicotine was collected on the first tube regardless of sampling rate examined except for one sample, while the amount of nicotine collected on the second tube was negligibly small.

### Stability of Nicotine Collected on Sampler Tube

After the injection of 5  $\mu$ l of *n*-propanol solution containing 100 ng of nicotine into the inlet portion of the sampler tube, nicotine-free air was passed through the tube at 40 or 100 ml/min for 8 hr to determine the stability of nicotine on the absorbent during a sampling period.

The results presented in Table 2 show that the recovery of nicotine after aeration is close to 100% regardless of aeration rate examined.

In a separate experiment a sampler tube containing 100 ng of nicotine was purged with nicotine-free air or nitrogen gas and then stored for 4 or 7 days at room temperature to determine the stability of nicotine on the absorbent during a storage period before GC analysis.

The results summarized in Table 3 show no significant difference in the recovery

2023380370

TABLE 2  
STABILITY OF NICOTINE ON SAMPLER TUBE FOR AERATION<sup>a</sup>

Aeration rate (ml/min)	Recovery after aeration (%)	
	Mean	SD
40	98.2	1.7
100	98.4	1.8

<sup>a</sup> Samples were aerated for 8 hr.

of nicotine among the samples stored for 4 or 7 days in different atmospheres. In any case the recovery is over 98% in average.

Such a high collection efficiency and stability of nicotine on the absorbent should be attributable to the acidic property of Unipori-S.

#### Desorption Efficiency of Nicotine from Sampler Tube

A mixture of 100 ng of nicotine and 400 ng of 7-MQ dissolved in 5 µl of n-propanol was injected into the sampler tube and then directly desorbed onto the GC column by heating. On the other hand, as a control experiment the same amount of the mixture was directly, i.e., not via sampler tube, injected into the GC column. In both experiments GC analyses were achieved under identical conditions. The recoveries of nicotine and 7-MQ thermally desorbed from the sampler tube were determined by comparing their peak areas with those of the control experiment.

The results are summarized in Table 4, which shows that the recoveries of both components increased to over 98% by the addition of a small amount of ammonia vapor in the initial period of thermal desorption. When no ammonia vapor was added, recoveries of nicotine and 7-MQ were reduced to about 85 and 95%, respectively. Such improvement of thermal desorption efficiency by addition of ammonia vapor is also attributable to the acidic property of Unipori-S.

TABLE 3  
STABILITY OF NICOTINE ON SAMPLER TUBE FOR STORAGE<sup>a</sup>

Storage period	Recovery after storage (%)	
	Mean	SD
Days in air		
4	98.9	1.4
7	99.1	2.8
Days in N <sub>2</sub>		
4	98.9	2.7
7	99.6	1.5

<sup>a</sup> Samples were stored at room temperature.

THE

Influe

Thr  
pollut  
differe  
a forc  
sampl  
obtain  
relativ  
was le

Tabl  
indepe  
larger  
tube i  
menta  
Howe  
erally  
should  
stream

Sa

2023380371

• THE

TABLE 4  
THERMAL DESORPTION EFFICIENCIES OF NICOTINE AND 7-METHYLQUINOLINE FROM SAMPLER TUBE

Addition of ammonia vapor to carrier gas	Thermal desorption efficiency (mean $\pm$ SD, %)	
	Nicotine	7-Methylquinoline
Yes	98.1 $\pm$ 4.2	99.1 $\pm$ 2.9
No	83.1 $\pm$ 8.3	94.5 $\pm$ 2.8

spheres. In  
absorbent:

5  $\mu$ l of n-  
d onto the  
the same  
d into the  
identical  
from the  
use of the

es of both  
ammonia  
apor was  
and 95%  
dition of

#### Influence of Sampling Rate on Measured Value of Nicotine Concentration

Three personal nicotine monitors were placed in the center of a closed chamber polluted with tobacco smoke. Nicotine was collected simultaneously at three different sampling rates, 40, 100 and 200 ml/min, for 15 min either with or without a forced wind from an electric fan. The amount of nicotine collected on each sampler tube was measured and corrected for respective sampling volume to obtain the nicotine concentration. The results shown in Table 5 are expressed as relative concentration, where the value obtained at a sampling rate of 40 ml/min was taken as 100.

Table 5 clearly shows that the measured value of nicotine concentration is independent of sampling rate whether there is a forced wind or not. For particles larger than 1  $\mu$ m, the concentration in the sampling stream entering the sampling tube is dependent on the velocity ratio of the sampling stream to the environmental stream, when there is a velocity difference between the two streams (7). However, since tobacco smoke particles suspended in environmental air is generally smaller than 0.5  $\mu$ m (10), the nicotine concentration in the sampling stream should be essentially independent of the sampling rate and the environmental stream, as demonstrated in Table 5.

TABLE 5  
EFFECT OF SAMPLING RATE ON MEASURED VALUE OF NICOTINE CONCENTRATION\*

Sampling rate (ml/min)	Run 1	Run 2	Run 3	Run 4	Ave.
(A) Without a forced wind					
40	100	100	100	100	100
100	103.0	94.1	103.2	98.3	99.6
200	104.2	95.4	102.1	101.8	100.9
(B) With a forced wind					
40	100	100	100	100	100
100	101.6	94.8	101.1	109.0	99.4
200	100.6	92.2	98.3	98.2	97.3

\* The value obtained at a sampling rate of 40 ml/min was taken as 100.

2023380372

### *Changes in Ambient Nicotine and Particulate Matter Concentrations*

An example of changes in concentrations of nicotine and particulate matter measured at a fixed station in a ventilated office is illustrated in Fig. 3. Particulate matter was monitored continuously with a respirable aerosol monitor (17) and the average value in 15 min was plotted, while nicotine was collected at 30-min intervals at a sampling rate of 200 ml/min. The profile of change in nicotine concentration was fairly comparable to that of particulate matter. Nicotine and particulate matter reached maximum concentration at the beginning of office hours.

### *Advantages of Nicotine Personal Monitor*

The range of accurate measurement of nicotine was from 5 to 500 ng per sampler tube. For example, if air samples are withdrawn at a rate of 40 ml/min for 1 hr, the personal monitor is applicable to the measurement of ambient nicotine ranging in concentration from 2 to 200  $\mu\text{g}/\text{m}^3$ . This sensitivity is about 10–100 times as high as that of previous methods (3, 18, 21) utilizing wet processes. Of course, sampling rate and/or sampling time should be changed according to the concentration of ambient nicotine. The other principal advantages of the monitor can be summarized as follows.

- (1) The monitor is lightweight, compact and pocketable.
- (2) The sampler tube can be reused after desorption of a test sample.
- (3) Direct thermal desorption of nicotine from the sampler tube onto the GC column simplifies and speeds up the analytical processes.
- (4) Since no pretreatment of analytical sample, except for the addition of 7-

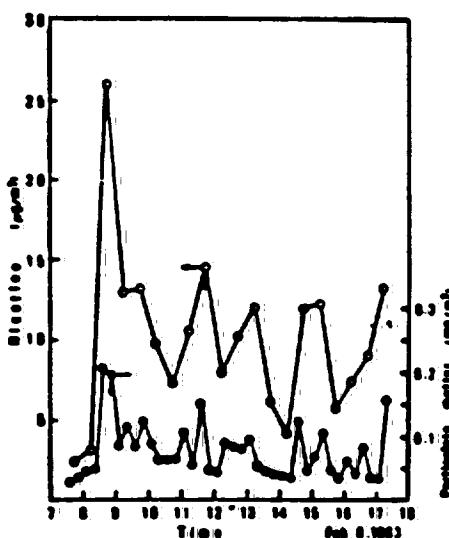


FIG. 3. Typical profiles of changes in concentrations of ambient nicotine and particulate matter. O, Nicotine; ●, Particulate matter.

MQ. is in  
nation by

### Evaluation

Using  
in various  
Table 6.  
estimated  
hr. "Equ  
resents th  
These va  
by the ni  
mg.

The co  
to be in  
examined  
smokers:  
40  $\mu\text{g}/\text{hr}$   
about 0.0  
passively.  
Yet, even  
hr of nic  
in one ho

Weber  
average in

### Loca

Office A
Office B
Laboratory
5 conference
3 hours
Hospital (1)
4 hotel (1)
7 tea room
3 restaurant
3 student c.
3 bus and r.
waiting to
4 cars
8 trains
7 airplanes
domestic

• Response  
• Amount

2023380373

late matter  
Particulate  
17) and the  
30-min in-  
-toxine con-  
-e and par-  
-ice hours.

: per sam-  
-min for 1  
1 nicotine  
- 10-100  
- > 4. Of  
ng. In the  
: monitor

the GC  
on of 7-

MQ. is involved before GC analysis, the sample is free from possible contamination by nicotine from glassware, solvent, etc.

#### Evaluation of Level of Passive Smoking in Various Living Places

Using the personal monitor attached to a nonsmoker, nicotine concentrations in various living environments were measured. The results are summarized in Table 6. "Amount of nicotine inhaled per hour" shown in the fifth column was estimated by multiplying nicotine concentration by respiration volume, 0.48 m<sup>3</sup>/hr. "Equivalent cigarettes smoked per hour" shown in the seventh column represents the level of passive smoking based on the amount of nicotine inhaled. These values were obtained by dividing "amount of nicotine inhaled per hour" by the nicotine amount inhaled by active smoking of one ordinary cigarette, 0.9 mg.

The concentration of ambient nicotine in various living environments was found to be in the range of 1.8 (in a laboratory) to 83 µg/m<sup>3</sup> (in a car) as far as we examined the matter. Therefore, the amounts of nicotine inhaled by passive smokers in their living environments could be estimated to be in the range of 0.9-40 µg/hr. These amounts are equivalent to those inhaled by active smoking of about 0.001-0.044 ordinary cigarettes in one hour. Average amounts of nicotine passively inhaled in cars, tea rooms, and conference rooms exceeded 15 µg/hr. Yet, even in these instances, passive smokers will rarely inhale more than 45 µg/hr of nicotine, corresponding to active smoking of about 0.05 ordinary cigarettes in one hour.

Weber and Fisher (20) found very low levels of nicotine of 0.9 ± 1.9 µg/m<sup>3</sup> on average in various workrooms. Hinds and First (8) measured nicotine concentra-

TABLE 6  
PERSONAL EXPOSURE TO AMBIENT NICOTINE IN VARIOUS LIVING ENVIRONMENTS

Location	No. of samples	Nicotine concentration (µg/m <sup>3</sup> )		Amount of nicotine inhaled (µg/hr)*		Equivalent cigarettes smoked (cigarettes/hr) <sup>†</sup>	
		Range	Average	Range	Average	Range	Average
Office A	4	9.34-31.57	19.44	4.5-13.2	9.3	0.0050-0.0168	0.0104
Office B	6	14.60-36.00	22.15	7.0-12.5	10.6	0.0076-0.0199	0.0118
Laboratory	8	1.76-9.64	5.00	0.8-4.6	2.8	0.0009-0.0051	0.0031
5 conference rooms	5	16.54-53.01	28.73	7.9-25.4	10.6	0.0083-0.0283	0.0206
3 houses	5	7.61-14.80	11.16	3.7-7.80	5.4	0.0041-0.0078	0.0060
Hospital lobby	7	1.09-5.02	2.98	0.9-2.4	1.4	0.0010-0.0027	0.0016
4 hotel lobbies	5	5.45-18.06	11.18	2.6-8.7	5.4	0.0029-0.0096	0.0066
7 tea rooms	12	15.10-60.09	33.41	7.2-29.2	16.0	0.0081-0.0325	0.0178
5 restaurants	8	7.09-27.81	14.76	3.4-13.3	7.1	0.0038-0.0148	0.0079
3 student cafeterias	6	11.59-42.16	26.42	5.6-30.2	12.7	0.0042-0.0225	0.0141
3 bus and railway waiting rooms	6	10.05-36.43	19.07	4.8-17.5	9.2	0.0054-0.0194	0.0102
4 cars	4	7.73-23.13	17.71	3.7-39.9	22.9	0.0041-0.0443	0.0254
8 trains	8	8.64-26.14	16.42	4.1-32.5	7.9	0.0046-0.0139	0.0088
7 airplanes (domestic airline)	7	6.28-28.78	15.18	3.8-13.8	7.3	0.0033-0.0153	0.0081

\* Respiration volume was estimated to be 0 liters/min.

† Amount of nicotine in main stream smoke was estimated to be 0.9 mg/cig.

2023380374

tions ranging from 1.0 to 10.3  $\mu\text{g}/\text{m}^3$  in various public places and estimated that the nonsmoker inhales tobacco smoke equivalent to active smoking of 0.001–0.01 filter cigarettes in one hour. Similarly Badre *et al.* (2) found that indoor nicotine concentrations were mostly less than 50  $\mu\text{g}/\text{m}^3$ .

The results shown in Table 6 are broadly consistent with those of Hinds and First, and Badre *et al.* Thus, the amount of tobacco smoke inhaled should be very much smaller for passive smokers than for active smokers, as previously pointed out by Klosterkötter and Gono (11).

Hugod *et al.* (9) demonstrated that nicotine concentration exceeded 100  $\mu\text{g}/\text{m}^3$  in a closed, unventilated smoky room in which CO concentration was held at a high constant level of 20 ppm by intermittent addition of freshly generated smoke. According to their estimates, at even such a significantly higher level, passive smokers must spend 50 hours in the room to inhale the same amount of nicotine as is inhaled by the active smoking of one cigarette. Asano (1) estimated that a nonsmoker exposed experimentally to tobacco smoke produced by 10 cigarettes per hour in a poorly ventilated room will have blood COHb level equivalent to smoking of one cigarette in one hour. In both experiments, however, smoke concentration is unrealistically high and will not represent the level that nonsmokers usually encounter in their living places.

#### REFERENCES

1. Asano, M. (1977). Passive smoking—Its environment and effects on health. *Igaku no Ayumi* 103, 479–499.
2. Badre, R., Guillerm, R., Abrau, N., Bourdin, M., and Dumas, C. (1978). Pollution atmosphérique par la fumée de tabac. *Ann. Pharm. Fr.* 36, 443–452.
3. Crouse, W. E., Johnson, L. F., and Marmor, B. S. (1980). A convenient method for the determination of ambient nicotine. *Brit. Tabakforsch.* 10, 111–113.
4. Feyerabend, C., Higenbottam, T., and Russell, M. A. H. (1982). Nicotine concentrations in urine and saliva of smokers and nonsmokers. *Brit. Med. J.* 284, 1002–1004.
5. Fischer, T., Weber, A., and Grandjean, E. (1978). Luftverunreinigung durch Tabakrauch in Gaststätten. *Int. Arch. Occup. Environ. Health* 41, 267–280.
6. Follett, D., Benowitz, N. L., and Becker, C. E. (1983). Passive absorption of nicotine in airline flight attendants. *N. Engl. J. Med.* 308, 1105.
7. Friedlander, S. K. (1977). "Smoke, Dust and Haze—Fundamentals of Aerosol Behavior," pp. 147–149. Wiley, New York.
8. Hinds, W. C., and First, M. W. (1975). Concentrations of nicotine and tobacco smoke in public places. *N. Engl. J. Med.* 292, 844–845.
9. Hugod, C., Hawkins, L. H., and Astrup, P. (1978). Exposure of passive smokers to tobacco smoke constituents. *Int. Arch. Occup. Environ. Health* 42, 21–29.
10. Ishizu, Y., Ochiai, K., and Tomita, H. (1982). Size distribution of cigarette smoke particles in a room. In "Proceedings, 6th International Symposium on Contamination Control," pp. 267–272. Japan Air Cleaning Assoc., Tokyo.
11. Klosterkötter, W., and Gono, E. (1976). Zum Problem des Passivrauchens. *Zentralbl. Bakteriol. Parasitenkd. Infektionskr. Hyg. Abt. I: Orig. Reihe B* 162, 51–69.
12. Matsuki, H., Onaka, F., Kasuga, N., and Sugita, M. (1981). Epidemiological study on the effects of smoking and air pollution using urinary hydroxyproline-creatinine ratio on the healthy schoolchildren and adults. *Japan. J. Public Health* 28, 505–515.
13. Muranaka, H., Tamada, T., Higashi, E., Itani, S., and Morimoto, M. (1981). Microdetermination of nicotine and its metabolites in biological fluid—Effect of smoking on plasma nicotine, plasma cotinine and other biochemical variables in human being. *Japan. J. Clin. Chem.* 10, 127–135.

14.  
15.  
16.  
17.  
18.  
19.  
20.  
21.

2023380375

- ated that  
001-0.01  
nicotine
- inds and  
ould be  
viously:  
  
10 µg/m<sup>3</sup>  
held at a  
smoke.  
passive  
zincine  
d that  
gargles  
able to  
smoke  
at non-
14. Oshinsky, S. J. (1982). Is smoker/non-smoker segregation effective in reducing passive inhalation among nonsmokers? *Amer. J. Public Health* 72, 737-739.
15. Russell, M. A. H., Cole, P. V., and Brown, E. (1973). Absorption by nonsmokers of carbon monoxide from room air polluted by tobacco smoke. *Lancet* 1, 567-579.
16. Russell, M. A. H., and Feyersabend, C. (1973) Blood and urinary nicotine in nonsmokers. *Lancet* 1, 179-181.
17. Sem, G. J., and Tsurabayashi, K. (1973). A new mass sensor for respirable dust measurement. *Amer. Ind. Hyg. Assoc. J.* 34, 791-800.
18. US Department of Health, Education and Welfare. (1977). "NIOSH Manual of Analytical Method," 2nd ed., Vol. 3, No. S-239.
19. Weber, A., Fischer, T., and Grandjean, E. (1979). Passive smoking in experimental and field conditions. *Environ. Res.* 20, 205-216.
20. Weber, A., and Fischer, T. (1980). Passive smoking at work. *Int. Arch. Occup. Environ. Health* 47, 209-221.
21. Yoshida, D. (1976). Measurement of nicotine in sidestream of tobacco smoke. *Sci. Pap. Cent. Res. Inst. Japan Tob. Sci. Public Corp.* No. 118, 203-206.

mi 103.  
phérique  
deter-  
in urine  
• Gestu-  
• airline  
or." pp.  
a public  
tobacco  
les in a  
p. 267-  
deterior.  
effects  
healthy  
infection  
plasma  
7-133.

2023380376

10/1/86  
J. C. Fischbeck Jr., Ph.D.  
Published by Springer-  
Verlag New York Inc.

## Estimation of personal exposure to ambient nicotine in daily environment

Motohiko Muramatsu<sup>1</sup>, Setsuko Umemura<sup>1</sup>, Junichi Fukui<sup>2</sup>, Tatsuo Arai<sup>2</sup>,  
and Shiro Kira<sup>2</sup>

<sup>1</sup>Central Research Institute, Japan Tobacco Inc., 6-2 Umegaoka, Midori-ku, Yokohama,  
Kanagawa 227

<sup>2</sup>Jichi Medical School, 3311-1 Yakushiji, Minamikawachi, Kawachi, Tochigi 329-04, Japan

**Summary.** To evaluate the actual exposure level of nonsmokers to environmental tobacco smoke (ETS) in their daily life, the exposure level of ambient nicotine was measured with a nicotine personal monitor carried by a nonsmoker. Average exposure levels of nicotine, even in such smoky places as cars, coffee shops and pubs, were less than 45 µg/m<sup>3</sup>. As a result of all-day monitoring, the highest amount of nicotine inhaled in a day was estimated, in this study, to be up to 310 µg, equivalent to actively smoking 0.3; ordinary cigarettes.

**Key words:** Passive smoking – Nicotine personal monitor – Environmental tobacco smoke (ETS) – Exposure level

### Introduction

Several epidemiological studies have suggested a relationship between passive smoking and an increased risk of lung cancer [12]. However, one major dispute about these studies is the lack of measurement of actual exposure to ETS [7, 10]. When studying the health effects of passive smoking, it is important to determine the actual exposure level of nonsmokers to ETS in daily life.

The exposure level to ETS has previously been evaluated by measuring concentration of such constituents as nicotine, cotinine and COHb (e.g., 4–6, 8) in body fluids. Meanwhile, the authors have recently developed a convenient personal nicotine monitor to estimate the exposure level of nonsmokers to ETS in the living places [9]. In the present work, by using the personal monitor carried by nonsmokers, exposure levels of nicotine in various daily environments and daily lives were measured.

### Materials and methods

The personal monitor consists of a sampler tube (Pyrex glass; 12-cm-long, 6-mm-i.d.) and a small sampling pump (about 34 ml/min; MD<sup>®</sup> Scientific Inc., Model 808) fitted with a means of

Offprints requests to: M. Muramatsu at the above address

2023380377

measuring sample volume. The personal monitor can be carried conveniently by a person throughout a sampling period. The sampler tube contains 450 mg of Uniport-S (60 to 80 mesh; Gas-kuro Kogyo Co.) coated with 4 wt% of silicone OV-17 as a nicotine absorbent. Prior to the coating, the acidity of Uniport-S was adjusted with KOH to 30 to 70  $\mu\text{mol/g}$  when calculated according to the n-butylamine titration method [11] using methyl red as an indicator. The sampler tube packed with the absorbent was aged at 310°C for 15 h by passing nitrogen gas through the tube at 40 ml/min.

Ambient nicotine was collected on the sampler tube by drawing air through the tube at a flow rate of 40 ml/min for a period of 1 to 8 h. After collection, 5  $\mu\text{l}$  of n-propanol solution containing 400 ng of 7-methylquinoline (7-MQ) was injected into the sampler tube as an internal standard. Then the sampler tube was placed in a cylindrical furnace heated to 280°C and connected to a gas chromatograph (GC) with a nitrogen-sensitive detector. By passing the carrier gas through the sampler tube, collected nicotine and 7-MQ were desorbed and directly transferred onto the GC column (2 m × 3 mm i.d. glass column) packed with Chromosorb W (AW-DMCS, 30 to 60 mesh) coated with 10 wt% PEG-20M and 2 wt% KOH. The thermal desorption was allowed to continue for 8 min, while the column temperature was held at 70°C in order to trap and concentrate both nicotine and 7-MQ on the top of the column. During the first 3 min of the thermal desorption, a small amount of ammonia vapor was added three times into the carrier gas by bubbling the carrier gas through 35 wt% ammonia water for 6 s, once per min. The addition of ammonia vapor is effective on the thermal desorption of nicotine and 7-MQ against the acidic property of Uniport-S. After the thermal desorption time, the column temperature was programmed to 185°C at 46°C/min and maintained at 185°C until completion of elution.

Both the collection and desorption efficiencies of nicotine were nearly 100% [9].

### Results and discussion

Figure 1 shows the average and standard deviation of personal exposure to ambient nicotine in various places. The values in the offices, households and pubs are an average from an 8-h sampling period, and the remainder are from a 1-h sampling period. The "amount of nicotine inhaled" shown in this figure was estimated by multiplying the nicotine concentration by a respiration volume of 0.48  $\text{m}^3/\text{h}$ . Then the "equivalent cigarettes smoked", which represent the level of passive smoking, can be obtained by dividing the "amount of nicotine inhaled" by the nicotine amount (1 mg) inhaled through active smoking from one ordinary cigarette.

The average exposure level of nicotine in three ventilated offices, A, B and C, was in the range of 5.9 to 19.8  $\mu\text{g}/\text{m}^3$ . The nicotine inhaled is estimated to be in the range of about 2.8 to 9.5  $\mu\text{g}/\text{h}$ . This value was calculated to be equivalent to the amount of nicotine inhaled through active smoking from 0.003 to 0.010 ordinary cigarettes/h.

Figure 2 shows the exposure levels measured over one week for three subjects, a, b and c at Office C with 72  $\text{m}^2$  of floor space. They were exposed to 3.0 to 10.2  $\mu\text{g}/\text{m}^3$  of nicotine in their ventilated office, where 28 to 48 cigarettes had been smoked daily. The exposure level of Subject a always showed the highest level, indicating that the actual exposure to ETS may differ with each subject, even in a small office.

Exposure levels in coffee shops, pubs and cars, naturally influenced by the number of cigarettes smoked and the ventilation conditions, showed relatively high values (Fig. 1). Average exposure level of nicotine in such places was in the

2023380378

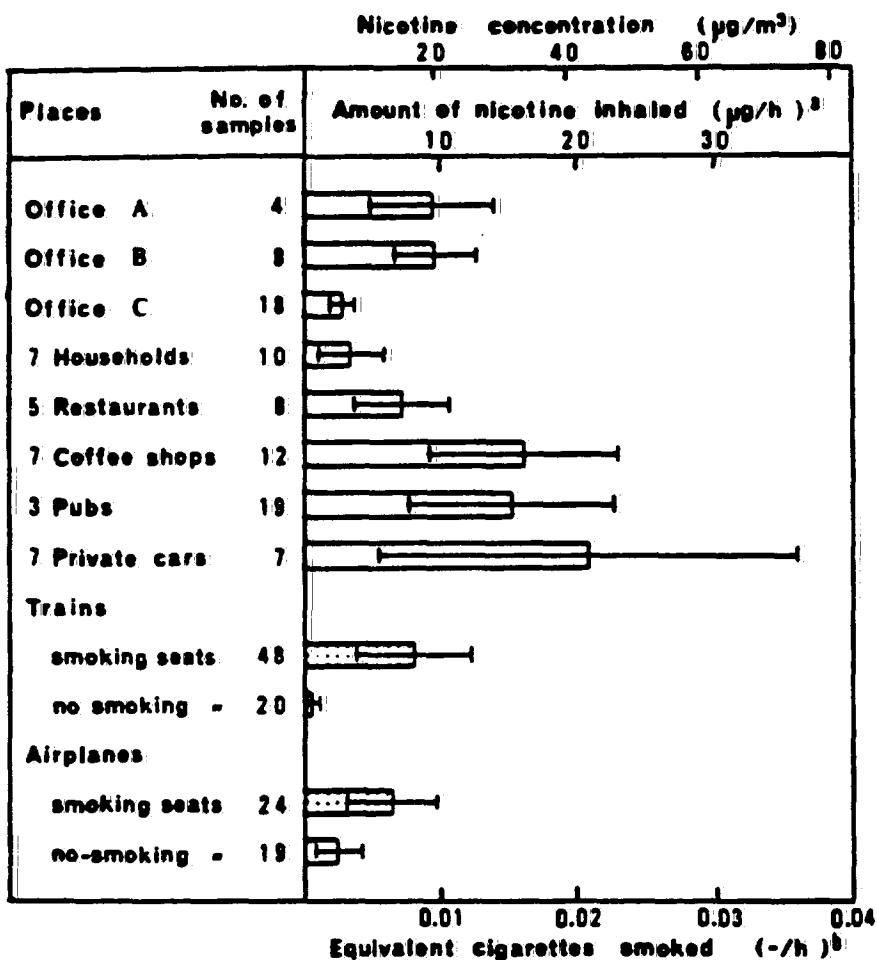


Fig.1. Personal exposure to ambient nicotine in various environments. (a) Respiration volume was estimated to be 8 l/min; (b) Amount of nicotine in main stream smoke was estimated to be 1 mg/cig.

range of 31.5 to 43.2  $\mu\text{g}/\text{m}^3$ . However, even in these instances, a nonsmoker does not inhale more than 50  $\mu\text{g}/\text{h}$  of nicotine, equivalent to active smoking of about 0.05 cigarettes/h.

The average exposure levels in the smoking seats and the no-smoking seats of trains and airplanes were 16.7 and 1.3, and 13.5 and 5.3  $\mu\text{g}/\text{m}^3$ , respectively (Fig. 1). The highest nicotine exposure levels in the smoking seats of trains and airplanes were 48.6 and 28.8  $\mu\text{g}/\text{m}^3$ , respectively. However, these values were only equivalent to the amount of nicotine inhaled through active smoking from 0.023 and 0.014 cigarettes/h.

The exposure level to ETS depends on many factors, including room size and time of day. Further, people do not live in the same environment for 24 h.

2023380379

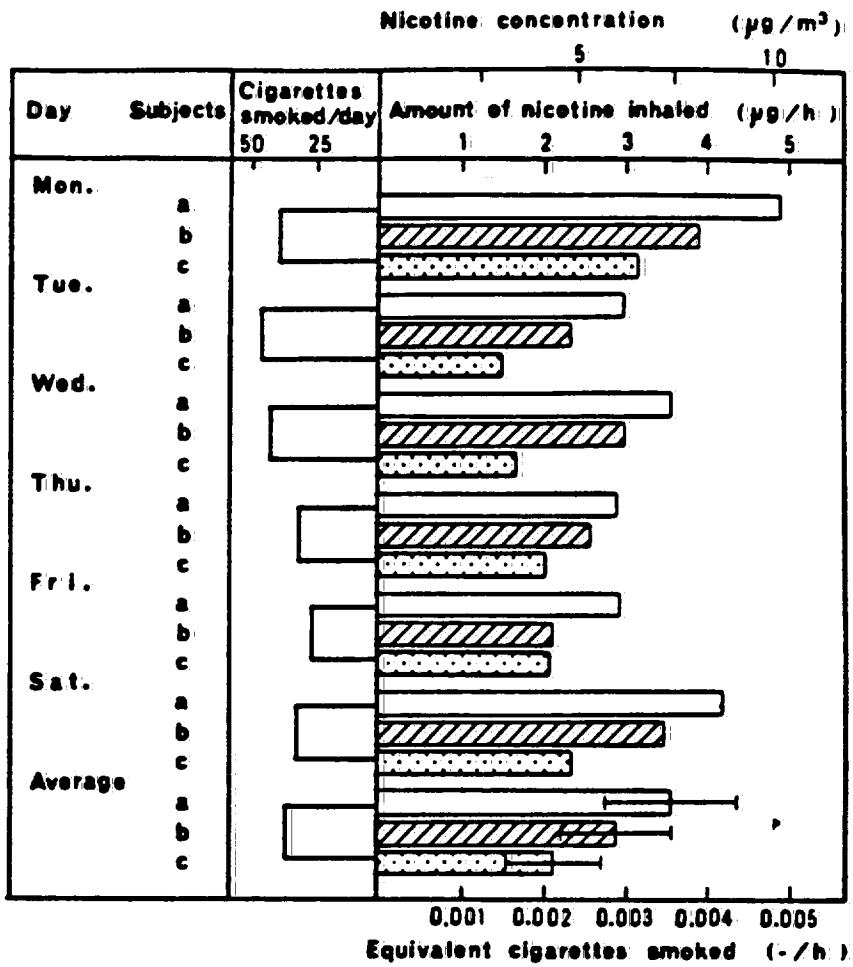


Fig. 2. Personal exposure of office workers to ambient nicotine in their office C

so the exposure level will differ with the behavior of individuals throughout the day.

Figure 3 shows the results of all-day monitoring of nicotine exposure for the nonsmoking subjects with or without an occupation in two cases where their families include and do not include a smoker. In this measurement, the sampler tube was exchanged every 8 h and the exposure level was monitored continuously for 1 to 6 d.

Nicotine intake is particularly high in subjects who are exposed to ETS at the workplace and at home. Some exposure to nicotine was also observed for many subjects without a smoker in their family. These results indicate that non-smokers without a smoker in their family are also exposed to ETS when a

2023380380

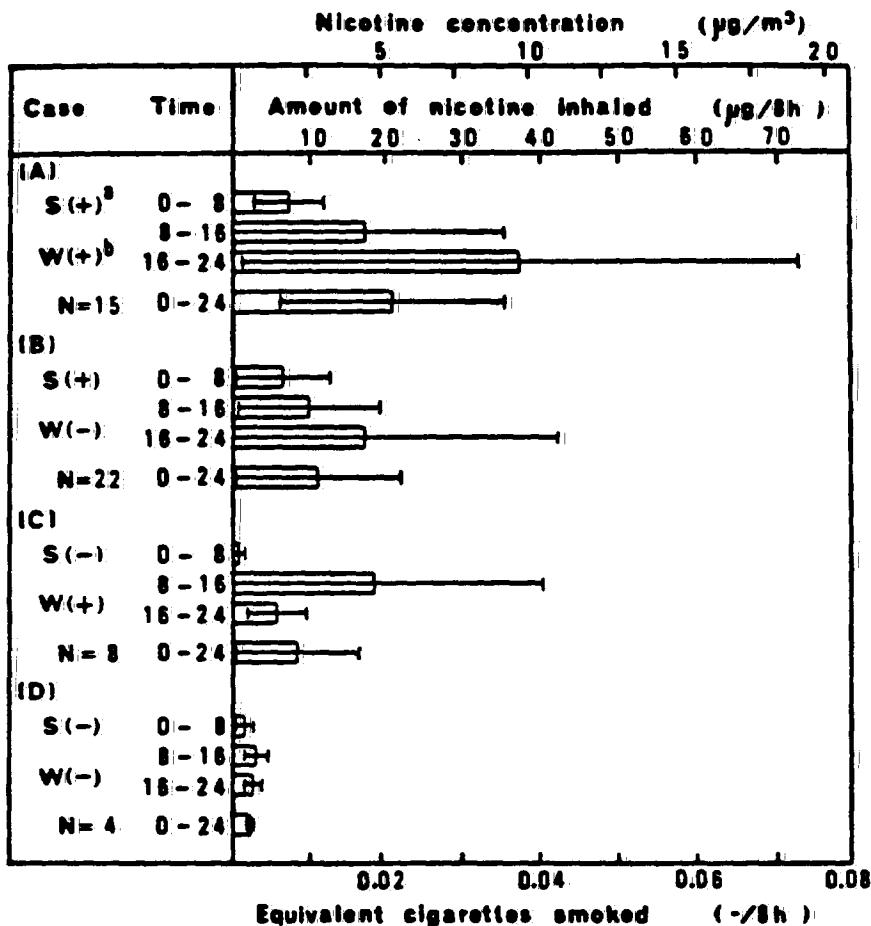


Fig. 3. Personal exposure to ambient nicotine over a period of 24 h in daily life. (a) S(+): with smoker in family. S(-): without smoker in family; (b) W(+): with occupation. W(-): without occupation

smoker visits, or when they go out. Therefore, it is impossible to evaluate their exposure to ETS based only on the smoking habits of their families.

In this all-day monitoring, the subject with the highest daily average of nicotine exposure was a housewife with a smoking husband. According to her report, she attended a party with him on that day. As a result, her daily average of exposure level amounted to  $27.3 \mu\text{g}/\text{m}^3$ , and the daily amount of nicotine inhaled was estimated to be up to  $310 \mu\text{g}$ , equivalent to actively smoking 0.31 cigarettes.

Hinds and First [3], Bardre et al. [1] and First [2] reported that nicotine concentrations in various public places were in the range of 1 to 10.3, 20 to 50 and 2.7 to  $30 \mu\text{g}/\text{m}^3$ , respectively. Our result is consistent with theirs. Thus, the nicotine level in daily environments will rarely exceed  $100 \mu\text{g}/\text{m}^3$ . If a man stays

2023380381

in a room with a 100 µg/m<sup>3</sup> nicotine level for 1 h, the amount of nicotine he will inhale is estimated only to be equivalent to that inhaled by actively smoking about 0.05 cigarettes.

Therefore, we can say that the amount of nicotine inhaled by a nonsmoker in his daily life is far smaller than that inhaled by a smoker through active smoking.

Nicotine is an excellent marker for ETS exposure because of its specificity for tobacco smoke. It is not necessarily clear, however, whether nicotine intake can provide the best quantitative estimation of the dose of ETS exposure or not. The composition of the sidestream smoke differs widely from that of mainstream smoke. The inhalation patterns in passive and active smoking are not comparable. The concentration of ambient nicotine decreases somewhat rapidly compared to that of other constituents of tobacco smoke, especially in a closed room without any ventilation. Consequently, in future it will be necessary to study quantitatively the relationship between nicotine, a vapor and particulate phase components of interest in ETS and biological markers such as cotinine to utilize nicotine fully as a quantitative marker of ETS exposure.

### References

1. Badre R, Guillerm R, Abran N, Bourdin M, Dumas C (1978) Pollution atmosphérique par la fumée de tabac. Ann Pharmacol Fr 35:443-452
2. First MW (1984) Environmental tobacco smoke measurement: retrospect and prospect. Eur J Resp Dis [Suppl 133] 65:9-16
3. Hinds WC, First MW (1975) Concentration of nicotine and tobacco smoke in public places. New Engl J Med 292:844-845
4. Hoffmann D, Haley NJ, Adams JD, Brunnemann KD (1984) Tobacco sidestream smoke: uptake by nonsmokers. Prev Med 13:608-617
5. Jarvis MJ, Russell MAH, Feyerabend C (1983) Absorption of nicotine and carbon monoxide from passive smoking under natural conditions of exposure. Thorax 38:829-833
6. Jarvis MJ, Russell MAH, Feyerabend C, Eiser JR, Morgan M, Gammie P, Gray EM (1985) Passive exposure to tobacco smoke: Saliva cotinine concentrations in a representative population sample of nonsmoking school children. Br Med J 291:927-929
7. Lehnert G (Chairman), Garfinkel L, Hirayama T, Schmidl D, Überla K, Wynder EL, Lee PN (1984) Roundtable discussion. Prev Med 13:730-746
8. Matsukura S, Taminato T, Kitano T, Seino Y, Hamada H, Uchihashi M, Nakajima H, Hirata Y (1984) Effect of environmental tobacco smoke on urinary cotinine excretion in nonsmokers. Evidence for passive smoking. New Engl J Med 311:828-832
9. Muramatsu M, Umemura S, Okada T, Tomita H (1984) Estimation of personal exposure to tobacco smoke with a newly developed nicotine personal monitor. Environ Res 35:218-227
10. Rylander R (1984) Environmental tobacco smoke and lung cancer. Eur J Resp Dis [Suppl 133] 65:127-133
11. Tamek MW (1950) Chemistry of the surface and the activity of alumina-silica cracking catalyst. Disc Faraday Sci 8:270-276
12. US Department of Health and Human Services, Public Health Service (1986) A report of the Surgeon General. The health consequences of involuntary smoking. DHHS Publication No. (CDC) 87-8398, pp 66-90

Received January 29 / Accepted May 19, 1987

2023380382

# A Thermal Desorption Method for the Determination of Nicotine in Indoor Environments

Cyril V. Thompson,\* Roger A. Jenkins, and Cecil E. Higgins

Organic Chemistry Section, Analytical Chemistry Division, Oak Ridge National Laboratory, Oak Ridge, Tennessee 37831

**NOTICE**  
This material may be  
protected by copyright  
law (Title 17 U.S. Code)

Nicotine, the major, unique component of the gas phase of environmental tobacco smoke (ETS), has been employed as a marker for estimating exposure to ETS. A personal monitoring system for the determination of exposure to nicotine has been developed. The system consists of a sampling cartridge packed with 200 mg of Tenax GC and a small, constant-flow, personal sampling pump. After sampling, the cartridges are analyzed by triethylamine-assisted thermal desorption gas chromatography with nitrogen-selective detection. Collection and desorption efficiencies for the cartridges have been determined. The system has been evaluated in controlled-atmosphere chambers, and applied in a variety of work sites, and in 36 restaurants, where measured concentrations of nicotine ranged from 0.5 to 37.2  $\mu\text{g}/\text{m}^3$ .

## Introduction

One of the major public health concerns of the 1980s has been indoor air pollution and its effects on the individual. Environmental tobacco smoke (ETS), which is the diluted and aged mixture of sidestream smoke emanating from the smoldering cigarette and mainstream smoke exhaled by the smoker, represents a potentially significant contribution to this pollution. Concentrations of ETS respirable suspended particulates (RSP) have been reported to range from 0 to 700  $\mu\text{g}/\text{m}^3$  in indoor environments (1). A number of procedures have been applied for estimating ETS concentrations based on the measurements of concentrations of particular ETS constituents, such as CO (2-5), oxides of nitrogen ( $\text{NO}_x$ ) (3-5), and particulate matter (4-9). However, these constituents of tobacco smoke are also the products of other combustion processes, an aspect that limits their utility as markers for estimating ETS levels, especially in complex atmospheres such as those existing in indoor environments. Estimates of personal exposure to ETS have been made by measuring carboxyhemoglobin ( $\text{COHb}$ ) (10, 11), urinary hydroxyproline (HOP) (12), and nicotine and cotinine (a metabolite of nicotine) in the blood, urine, and saliva (10, 11, 13).

Several methods have been developed for determining nicotine concentrations at fixed sampling locations in industrial settings. The NIOSH method for nicotine utilizes a resin-filled cartridge (XAD-2) with a personal sampling pump for collection of samples followed by solvent extraction and analysis by gas chromatography (14). However, this method's 300  $\mu\text{g}/\text{m}^3$  limit-of-detection (LOD) makes it unsuitable for measuring ETS because associated concentrations of nicotine are well below this LOD. Another industrial method, also limited by its relatively high LOD (40  $\mu\text{g}/\text{m}^3$ ), collects nicotine in a series of water-filled bubblers (15).

Williams et al. (16) have reported a method using a cold Petri dish as the means for collecting nicotine. Although the reported nicotine concentration range associated with the method was low enough to be applicable for measuring ETS, the method had several deficiencies that would severely limit its value (17). Other methods reported in the literature detail the use of untreated glass fiber filters (4).

or diffusion desuder tubes (18) for collection of ambient nicotine.

The development and testing of a number of personal monitoring systems that measure individual exposures to ETS as determined by ambient nicotine concentrations have been reported recently in the literature. Solvent desorption based systems include personal sampling pumps coupled with commercially available XAD-4 cartridges (19-21) and  $\text{NaHSO}_4$ -treated, Teflon-coated glass fiber filters (22), and a passive sampling system utilizing the treated filters (23). The limitation of using solvent extraction of samples is that only a small fraction of the analyte is actually analyzed. This necessarily raises the theoretical LOD for such methods relative to those such as thermal desorption that use all of the acquired sample. Two thermal desorption based personal monitoring systems for nicotine have been reported, one by Proctor (24) that employs an unspecified adsorbent and analysis system and another by Muramatsu et al. (25, 26) that utilizes an ammonia purge of the sample cartridge during desorption into a gas chromatograph (GC). In initial laboratory evaluation studies, we found the experimental arrangement used by Muramatsu to be cumbersome and mechanically complex. In addition, we found that repeated exposures of the analytical column to the ammonia gas caused rapid deterioration of the column. Furthermore, the collection cartridge utilizes a packing, the support for which is a very unique diatomaceous earth, that is difficult to obtain in the United States.

This paper discusses the development, evaluation in controlled ETS atmospheres in chambers and offices, and field validation of a thermal desorption based personal monitoring system for nicotine using Tenax-GC as the adsorption material. Tenax is a poly(p-2,6-diphenyl-phenylene oxide) which is porous and stable up to 400 °C (27). It has a high affinity for semivolatile organic compounds and can be used repeatedly. It has been used for a number of indoor air characterization studies (28, 29), and its performance characteristics are relatively well understood. The nitrogen-selective detector used in the analysis procedure affords improved sensitivity and selectivity over a conventional flame ionization system when assaying complex atmospheres and has been employed similarly by several investigators (19, 20). The experimental arrangement used for the field sampling is similar to that employed by other investigators. The analytical system lacks the mechanical complexities of the system developed by Muramatsu.

## Methods and Material

**Personal Monitoring Systems.** Air-sampling cartridges were 16-cm sections of  $1/4$ -in.-o.d. borosilicate glass tubing which were treated with  $\text{NH}_4\text{OH}$  (immersion in 15%  $\text{NH}_4\text{OH}$  overnight, followed by air drying) and then fire polished on both ends and packed with approximately 200 mg of Tenax GC, 35-60 mesh, acquired from Alltech Associates (Deerfield, IL). Before use, the packed cartridges were conditioned at 250 °C by attaching them to a manifold in the oven of a gas chromatograph and passing

2023380000

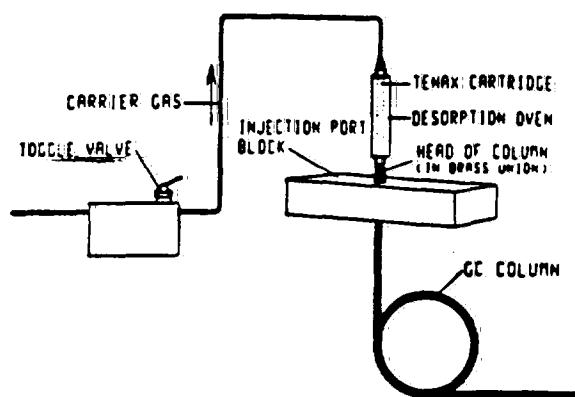


Figure 1. Schematic diagram of analytical system.

a stream of N<sub>2</sub> flowing at 40 mL/min through each cartridge for at least 2 h. After the cartridges had been cooled with continued N<sub>2</sub> flow, both ends were sealed with 1/4-in. plastic caps obtained from Alltech Associates. The cartridges could be reused by first washing them with 2-3 mL of methanol and thermally reconditioning them according to the procedure given above.

Alpha-2 personal sampling pumps, available from DuPont (Kennett Square, PA), were used for sample collection in most experiments (DuPont P-4000 pumps were used in a few initial chamber experiments) and were chosen for their light weight (410 g) and low noise level during operation. This latter feature was especially important for unobtrusive performance. For experiments performed in the chamber and work areas, in which occupants were aware of the sampling being conducted, pumps were connected to Tenax cartridges with a section of flexible tubing, and air from the area sampled was drawn through the cartridge. For sampling conducted in restaurants, in which the occupants were unaware of the sample collection, the pumps were worn on belt clips under jackets. A section of Tygon tubing was used to connect the pump to the Tenax cartridge, the latter being clipped to the inside lapel of the jacket so that the inlet end of the cartridge was within 25 cm of the mouth and nose of the individual conducting the sampling. All samples were collected for at least 1 h with the pump operating at a flow rate of 170 mL/min. Flow rates were checked with a bubble meter before and after sample acquisition. Immediately after completion of sampling, Swagelok 1/4-in. stainless steel end caps fitted with Teflon ferrules were placed on each end of the cartridge and tightened, and the cartridge was refrigerated at 3 °C until analysis.

**Analytical Method.** Nicotine containing solution standards were prepared by diluting redistilled nicotine (98%) obtained from Eastman Kodak (Rochester, NY) in ethyl acetate that contained 0.01% triethylamine (TEA). Internal standards employing quinoline were prepared by diluting quinoline in a solution of ethyl acetate/5% TEA. Fresh nicotine and quinoline standards were prepared every 15 days.

Analyses were performed with a Varian Model 3700 gas chromatograph equipped with a nitrogen/phosphorus detector (GC/NPD) and a 2 m × 2 mm i.d. glass column packed with 10% Carbowax 20M/2% KOH on 80-100 mesh Chromosorb W-AW (obtained from Alltech Associates). Flow rates were He (carrier gas) 40 mL/min, H<sub>2</sub> 4.5 mL/min, and air 175 mL/min. Temperature settings were injector and detector 250 °C and column oven initial temperature 70 °C for 8 min, programmed at a rate of 46 °C/min to 175 °C for 4 min. At these settings, nicotine elutes at 13.4 min and quinoline at 14.0 min.

In Figure 1 is portrayed a schematic diagram of the analytical experimental configuration. The system is designed to be mechanically simple and to minimize the opportunity for the nicotine vaporized from the Tenax trap to contact any materials prior to entering the analytical column. The carrier gas is directed through a toggle valve so that it can be interrupted when the Tenax cartridges are being changed. (When the system is not in use, a clean glass tube replaces the cartridge.) The analysis is performed by loosening the fittings at both ends of the desorption oven, inserting the cartridge, tightening the fittings, and resuming the carrier gas flow. Although this operation remains at operating temperature during this operation, the elapsed time for connecting the cartridge is less than 5 s. The desorption begins when the carrier gas is turned on.

Because the manner in which the analyte is introduced into the gas chromatograph affects the peak shape and ultimately the apparent quantity of analyte present in the aliquot, it was critical that the analyte in the calibration standards be introduced in a manner identical with that of those in the samples. To accomplish this, clean Tenax-filled cartridges were spiked with small aliquots of nicotine standard solutions (on the downstream end to simulate sample loading) by using a conventional 10-μL syringe. The volume of the aliquots ranged from 1.8 to 6 μL, depending on the desired amount of standard. Next, the cartridges were spiked with 5 μL of the ethyl acetate solution containing the quinoline internal standard, the TEA desorption modifier on the upstream end to facilitate desorption of the entire cartridge. In order to maintain direct comparability, this was the same quinoline/TEA solution that was added to the ETS samples. In the initial developmental work for the method, multipoint calibrations with nicotine standards were performed daily. For the field sampling, a calibration curve was generated from the desorption of nine sets of duplicate Tenax traps loaded with amounts of nicotine ranging from 1.5 to 700 ng and with 250 ng of quinoline internal standard prior to any sample analysis. The first set of standards was run in one random order and the second set of standards was run in a different random order. Daily standards of 3, 100, and 700 ng of nicotine were analyzed during sample analysis to ensure analytical control. Field blank cartridges were analyzed periodically.

Because the response of the nitrogen/phosphorus detector tended to be nonlinear at higher trap loadings (>1000 ng), data from the analyses were fitted to a second-order polynomial regression. In practice, there was no difference between first- and second-order regressions in the 0-700-ng concentration range. For example, the first- and second-order correlation coefficients ( $R^2$ ) for one calibration run were both 0.995, and for another, both were 0.997.

Response factors (RF) for all standards were calculated with the formula

$$RF = \left( \frac{\text{area counts nicotine}}{\text{area counts quinoline}} \right) \times \left( \frac{\text{concentration of quinoline}}{\text{concentration of nicotine}} \right) \quad (1)$$

Averages and standard deviations computed from the RF data were used to assess control of the method in day-to-day operation. If results for daily control standards were more than two standard deviations from the average for the calibration, the method was judged to be out of control, thus requiring recalibration. Control was observed

2023380384

throughout the analysis of the samples from the public areas.

**Test Atmospheres.** The initial experimental atmospheres for the development of the Tenax method were generated in two stainless steel chambers with volumes of 0.4 and 1.4 m<sup>3</sup> (obtained from Young and Bertke Co., Cincinnati, OH). Sidestream smoke from a 2R1 Kentucky Reference cigarette (procured from the University of Kentucky Tobacco and Health Research Institute, Lexington, KY), smoldering in a laminar flow smoke generation (30), was pulled into the smaller chamber at a rate of 30 L/min and diluted with an air flow of 250–1000 L/min, the exact rate depending on the concentration of ETS needed. Concentrations for this chamber ranged from 700 to 3500 µg/m<sup>3</sup> particulate matter (PM) and from 100 to 500 µg/m<sup>3</sup> nicotine. Low concentrations of ETS, 50–300 µg/m<sup>3</sup> PM and 10–70 µg/m<sup>3</sup> nicotine, were generated by diluting a portion of the atmosphere from the small chamber into that of the large chamber. Concentrations of particulate matter in the chambers were monitored with a TSI-5000 piezoelectric balance (acquired from TSI, St. Paul, MN) and an RAS-1 light-scattering sensor (purchased from GCA Instruments, Bedford, MA), which was modified in our laboratory to enhance its sensitivity. The nicotine and PM concentrations utilized for these experiments are much higher than what would be typically observed in real life situations and were used only to determine the potential utility and the upper analytical limits of the method. After development experiments involving the chamber were concluded, other experiments were conducted in an unoccupied office. ETS was produced by generating sidestream smoke from 1R4F Kentucky Reference cigarettes smoked (one 35-mL puff/min) on an ADL-II machine obtained from Arthur D. Little Co., Cambridge, MA). Mainstream smoke was collected in sealed Tedlar bags (acquired from SKC Inc., Eighty Four, PA), and ETS concentrations were varied by adjusting the smoking rate from 1 min of smoking (2-s puff, 58-s smolder) per min of elapsed time up to continuous cigarette smoking. PM levels were monitored with a TSI-5000 piezoelectric balance.

Additional laboratory evaluations of the method's performance were conducted in an 18-m<sup>3</sup> environmental chamber (31) used for ETS studies and located at the R. J. Reynolds Tobacco Company's facilities in Winston-Salem, NC. PM concentrations in that chamber were monitored with a TSI-5000 piezoelectric balance. Initial field evaluations were conducted in work areas, offices, common areas, and dining areas at Oak Ridge National Laboratory.

**Sampling Site Selection.** Field sampling was conducted in establishments that were both listed under the "Restaurant" heading in the Yellow Pages of the Knoxville, TN, telephone directory and located in the Knoxville, TN, Standard Metropolitan Statistical Area (SMSA) (Knox, Blount, and Anderson counties). Restaurant selection was conducted by assigning each restaurant a number and then choosing 43 out of the 419 restaurants with a random number generator. Three of these restaurants were eliminated because they had gone out of business, three because they were carry out only, and one because the personal safety of the sampling team was called into question. The remaining 36 were sampled, and for each sample, information was recorded regarding the number of smokers, the number of cigarettes, cigars, and pipes observed to have been smoked, the distance to the closest observed smoker, the type of meal served (lunch or dinner), crowd density, and restaurant volume. All of the information was recorded on a sampling data sheet during the

**Table I. Nicotine and Particulate Matter (PM) Concentrations Measured in 0.4- and 1.4-m<sup>3</sup> Stainless Steel Chambers\***

	Nicotine, µg/m <sup>3</sup>	PM, µg/m <sup>3</sup>	Nicotine/PM ratio
34	80	0.425	
42	83	0.506	
43	79	0.544	
44	74	0.595	
mean ± 1 SD <sup>a</sup>	40.8 ± 4.6	79.0 ± 3.7	0.518 ± 0.072
282	757	0.373	
263	667	0.394	
302	684	0.442	
291	659	0.442	
238	708	0.336	
224	684	0.328	
271	643	0.421	
mean ± 1 SD <sup>a</sup>	267 ± 23	696 ± 36	0.391 ± 0.047

\* Air changes per hour (ACH) ranged from 21 to 150. <sup>a</sup>SD, standard deviation.

time of sampling. A unique sample number was assigned to each cartridge immediately following sampling. No attempt was made to assess air exchange within the facility, as this would have compromised the unobtrusive nature of the sampling. Also, no determination of the number of smokers smoking at any one time or smoker turnover was made. In addition to the samples acquired in restaurants, two samples were acquired on a Saturday afternoon at each of three food courts in shopping malls.

### Results and Discussion

Results of initial experiments with Tenax cartridges, in which the responses to standard quantities of nicotine spiked on to the cartridges and subsequently desorbed were compared with those of the same sized aliquot directly injected on to the head of the GC column, showed evidence of incomplete desorption of nicotine, with up to 10% of the nicotine remaining on the cartridge. In order to enhance nicotine desorption, an internal standard solution was prepared that included 5% TEA. It has been found that addition of a strongly basic material such as TEA (19) or NH<sub>4</sub>OH (32) to nicotine standards prevents adsorption, by the glass of the container, of nicotine from solution. The base probably functions by displacing nicotine or other weaker bases from the adsorptive sites. Internal standard spikes thus contained about 200 µg of TEA, which, as a stronger base, displaced nicotine from acidic sites within the sampling cartridge or analysis train.

Experiments conducted in the 0.4- and 1.4-m<sup>3</sup> chambers were performed to determine the functional capabilities of the method and the nicotine collection efficiency. Table I gives the results from sampling of both dilute and concentrated simulated ETS environments in the large and small chambers, respectively. The ratios of nicotine to particulate matter in these experiments are substantially higher than what has been reported in typical indoor environments (33). This discrepancy was judged of little consequence since investigation of nicotine levels was the sole focus of the study. However, the consistency of the ratios is about ±15% or less, which was judged to be indicative of both a constant atmosphere in the chamber and consistent nicotine and particulate mass concentration determinations.

Experiments to determine sample volumes at which nicotine breakthrough became significant were conducted by placing two Tenax cartridges in series and sampling from simulated ETS environments in the chambers. Re-

2023380385

**Table II. Concentrations of Nicotine and Particulate Matter (PM) Measured in an Unoccupied Office**

trial no.	PM, μg/m <sup>3</sup>	nicotine, μg/m <sup>3</sup>	trial no.	PM, μg/m <sup>3</sup>	nicotine, μg/m <sup>3</sup>
1	14.6	1.8	6	59.6	8.2
2	23.2	1.9	7	113.0	21.7
3	28.2	2.4	8	115.4	27.3
4	15.2	3.7	9	257.0	48.3
5	58.6	4.8	10	248.8	49.0

**Table III. Results from Determination of Nicotine by the Texas Method in a Minimal Air Exchange Controlled Atmosphere Chamber**

run no.	PM, μg/m <sup>3</sup>	nicotine, μg/m <sup>3</sup>	run no.	PM, μg/m <sup>3</sup>	nicotine, μg/m <sup>3</sup>
1	55	2.5	4	62	4.1
2	14	1.8	5	16	2.1
3	103	6.0	6	128	6.5

\*N = 3 determinations.

sults indicated not more than 1% breakthrough for sample volumes ranging from 20 to 45 L and nicotine concentrations ranging from 70 to 250 μg/m<sup>3</sup>. At lower sample volumes, breakthrough percentages are expected to be correspondingly lower.

In Table II are listed the results from sampling of ETS in the unoccupied office. This range of nicotine and PM levels more closely approximated that which would be expected from sampling in public places. Proportionality between nicotine and particulate levels was particularly good in this experiment, with the correlation coefficient of 0.976 for a first-order regression analysis of these two parameters.

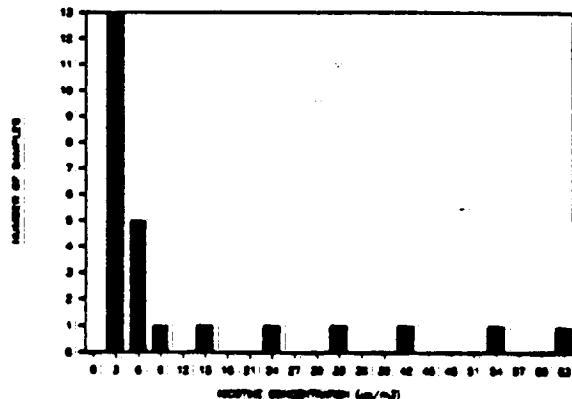
The limits of detection and quantitation were determined according to published guidelines (34). These are comparable to 3 and 10 times the standard deviation above the mean value of a series of field blanks, respectively. Signal response of the blanks (in microvolt seconds) was related to a series of calibration standards run within the lower quantitation region. According to these criteria, under the sampling conditions described above, the limit of detection was equivalent to 0.07 μg/m<sup>3</sup> nicotine, and the limit of quantitation was 0.17 μg/m<sup>3</sup>. This calculated level is in good agreement with experiences with sampling actual low-concentration ETS atmospheres in an office environment. These experiments indicated that within the range of 0.2–0.3 μg/m<sup>3</sup>, variation among multiple samples acquired near the same point in space became unacceptably large. Presumably, the effective limit of detection could be lowered by simply increasing the sampling duration.

In Table III are listed the results from sampling conducted in an 18-m<sup>3</sup> chamber at R. J. Reynolds. The purpose of these experiments was to determine the performance of the method in a chamber whose atmosphere had been well characterized in a number of studies (19, 31), especially at low nicotine concentrations, and to compare nicotine with ETS PM levels in a controlled environment that had been contaminated with ETS only. The nicotine levels sampled for this experiment are near the mean of the level determined in the field study (see below) but represent only a fraction of the range expected to be encountered during field sampling in general. For this and all the experiments where PM concentration data were available, mean ratios of nicotine to PM were calculated. The ratios obtained from experiments involving the 1.4- and 0.4-m<sup>3</sup> chambers were 0.52 ± 0.07 and 0.39 ± 0.05, respectively. The ratio for experiments performed in the

**Table IV. Nicotine Concentrations Measured at Selected Locations within Oak Ridge National Laboratory**

location	ambient nicotine level, μg/m <sup>3</sup>	location	ambient nicotine level, μg/m <sup>3</sup>
offices	4.2 ± 0.1*	common area	30.0 ± 0.9
	4.0 ± 3.5*		60.3 ± 2.1
	4.5 ± 0.5*		53.1 ± 2.8*
	6.7 ± 0.9		23.2 ± 2.9
	0.7 ± 1.0		39.7 ± 0.1
	1.1 ± 1.5		12.6 ± 1.1
	0.6 ± 0.8		3.8 ±
	0.6 ± 0.8		2.2 ±
	0.6 ± 0.9		1.0 ±
	0.3 ± 0.4		0.9 ± ..
dining area	4.4 ± 0.8		1.7 ± 0.8
	2.3*		0.8 ± 1.1
work area	2.0 ± 0.3		

\*N = 3 determinations. \*N = 1. All others, N = 2 determinations.



**Figure 2. Distribution of nicotine levels for work sites at ORNL. Note that nicotine concentrations listed are the maximums for the individual sites.**

unoccupied office was 0.16 ± 0.06, and for experiments conducted in the 18-m<sup>3</sup> chamber, 0.08 ± 0.04. The considerable differences among the ratios are expected in view of the differences in ETS levels, air handling methods, and air exchange rates and the theory that as ETS ages, nicotine tends to be adsorbed by the various surfaces present (18, 26). For the two smaller chambers, air exchange rates ranged from 21 to 150 ACH, allowing little opportunity for nicotine adsorption by the chamber walls. The office, with an air exchange of 5.4 ACH and nonrecirculated ventilation, exhibited lower nicotine/PM ratios, and the 18-m<sup>3</sup> chamber, with an air exchange rate of 0.05 ACH (35) — complete recirculation in a static system, abowed the levels of nicotine relative to the particulate levels. The data appear to support the above-mentioned theory.

Listed in Table IV are the results from samples collected at Oak Ridge National Laboratory facilities. The arithmetic mean and standard deviation of nicotine concentrations for all sample sites is 10.5 ± 17.2 μg/m<sup>3</sup>. However, the relatively high concentrations measured in the first common area (36.5 ± 18.1 μg/m<sup>3</sup>) influence the average disproportionately. As can be seen in Figure 2, the data appear to be distributed in a log normal pattern; thus, the geometric mean of 3.2 μg/m<sup>3</sup> (with 95% confidence boundaries of 1.8 and 6.0 μg/m<sup>3</sup>) may be more appropriate for this data set. For the lower nicotine level environments, there is considerable variation ( $\pm 100\%$ ) within duplicate samples taken near the same point in space. For the environments containing higher nicotine levels, the coefficient of variation within duplicate samples was usually about  $\pm 10\%$ .

2023380386

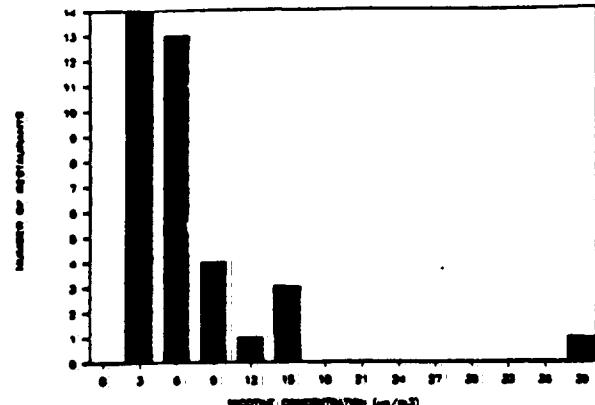


Figure 3. Distribution of nicotine resultant selected Knoxville, TN, area restaurants. Note that nicotine concentrations listed are the maximums for the individual cells.

Results from the field determinations of nicotine concentrations in restaurants are shown in Table V. Nicotine concentrations found in the restaurants ranged from 0.5 to 37.2  $\mu\text{g}/\text{m}^3$  with an arithmetic mean of  $5.4 \pm 6.4 \mu\text{g}/\text{m}^3$ . As in the treatment of the data from the in-house sampling, a plot of the distribution of the concentration data indicates that it fits a log normal, rather than Gaussian, pattern (Figure 3). The geometric mean of  $3.5 \mu\text{g}/\text{m}^3$ , with 95% confidence boundaries on the median of distribution of 2.5 and  $4.8 \mu\text{g}/\text{m}^3$ , is somewhat lower than the arithmetic mean but is still comparable to data results cited by the researchers listed below.

Muramatsu et al. (25) reported a range of 7.1–27.8  $\mu\text{g}/\text{m}^3$  nicotine with an average of  $14.8 \mu\text{g}/\text{m}^3$  for eight samples taken in five restaurants. Hinds and First (36) have reported an average of  $5.2 \mu\text{g}/\text{m}^3$  nicotine for four samples from restaurants. Oldaker et al. (21) reported a range of 0–24  $\mu\text{g}/\text{m}^3$  nicotine with an average of  $5 \mu\text{g}/\text{m}^3$  for 170 samples acquired in restaurants. For air samples taken in offices, Hammond et al. (22) have reported 3–48  $\mu\text{g}/\text{m}^3$  nicotine, while Muramatsu et al. (25) have reported 9–32 and 6–20  $\mu\text{g}/\text{m}^3$  nicotine (26). For 156 office samples, Oldaker et al. (21) reported an average of  $5 \mu\text{g}/\text{m}^3$  nicotine with a range of 0–70  $\mu\text{g}/\text{m}^3$ . Nicotine concentrations in public common areas such as lobbies and waiting rooms were reported to be 2–36  $\mu\text{g}/\text{m}^3$  by Muramatsu et al. (25) and 1–3  $\mu\text{g}/\text{m}^3$  by Hinds and First (36). For samples taken in the smoking sections of airplanes, Muramatsu et al. (26) have reported 14 and 6–29  $\mu\text{g}/\text{m}^3$  with an average of  $15 \mu\text{g}/\text{m}^3$  (25). Oldaker and Conrad (20) have reported 0–112  $\mu\text{g}/\text{m}^3$  nicotine, with an average of  $9 \mu\text{g}/\text{m}^3$  in airplane smoking sections, and 0–40  $\mu\text{g}/\text{m}^3$  nicotine, with an average of  $6 \mu\text{g}/\text{m}^3$  in nonsmoking sections.

Major factors likely to affect nicotine concentrations in a public location include the number of cigarettes smoked and the time required for smoking, the volume of the room, the proximity of smokers to the sample location, and the air exchange rate. Under the conditions of the field sampling validation for this study, not all of these parameters could be easily determined, nor were they necessary to assess the performance of the experimental personal monitor in a realistic situation. However, to assess the impact of the easily determined factors, the relationship between those factors and ambient nicotine concentrations were determined. The Pearson's correlation coefficients (*r*) for first-order regressions of nicotine concentration to number of cigarettes, number of smokers, restaurant volume, and distance to the closest smoker were computed to be 0.669, 0.783, 0.049, and -0.155, respectively. The significance levels (*p*) (an indicator of the probability of

Table V. Nicotine Levels in Restaurants

sample no.	cigarettes, no.	smokers, no.	nicotine concn., $\mu\text{g}/\text{m}^3$	closest smoker, ft
1	1	1	4.2	7
2	6	6	4.3	6
3	6	6	4.5	14
4	6	6	4.6	10
5	6	6	4.8	5
6	7	7	6.6	2
7	7	7	6.7	2
8	7	7	7.3	7
9	7	7	7.4	7
10	7	7	7.6	7
11	8	8	8.0	10
12	8	8	8.0	204
13	8	8	8.3	177
14	8	8	8.3	793
15	8	8	8.5	1666
16	8	8	12.6	640
17	8	8	13.6	272
18	8	8	37.2	8
19	—	—	—	—
20	—	—	—	—
21	—	—	—	—
22	—	—	—	—
23	—	—	—	—
24	—	—	—	—
25	—	—	—	—
26	—	—	—	—
27	—	—	—	—
28	—	—	—	—
29	—	—	—	—
30	—	—	—	—
31	—	—	—	—
32	—	—	—	—
33	—	—	—	—
34	—	—	—	—
35	—	—	—	—
36	—	—	—	—
37	—	—	—	—
38	—	—	—	—
39	—	—	—	—
40	—	—	—	—
41	—	—	—	—
42	—	—	—	—
43	—	—	—	—
44	—	—	—	—
45	—	—	—	—
46	—	—	—	—
47	—	—	—	—
48	—	—	—	—
49	—	—	—	—
50	—	—	—	—
51	—	—	—	—
52	—	—	—	—
53	—	—	—	—
54	—	—	—	—
55	—	—	—	—
56	—	—	—	—
57	—	—	—	—
58	—	—	—	—
59	—	—	—	—
60	—	—	—	—
61	—	—	—	—
62	—	—	—	—
63	—	—	—	—
64	—	—	—	—
65	—	—	—	—
66	—	—	—	—
67	—	—	—	—
68	—	—	—	—
69	—	—	—	—
70	—	—	—	—
71	—	—	—	—
72	—	—	—	—
73	—	—	—	—
74	—	—	—	—
75	—	—	—	—
76	—	—	—	—
77	—	—	—	—
78	—	—	—	—
79	—	—	—	—
80	—	—	—	—
81	—	—	—	—
82	—	—	—	—
83	—	—	—	—
84	—	—	—	—
85	—	—	—	—
86	—	—	—	—
87	—	—	—	—
88	—	—	—	—
89	—	—	—	—
90	—	—	—	—
91	—	—	—	—
92	—	—	—	—
93	—	—	—	—
94	—	—	—	—
95	—	—	—	—
96	—	—	—	—
97	—	—	—	—
98	—	—	—	—
99	—	—	—	—
100	—	—	—	—
101	—	—	—	—
102	—	—	—	—
103	—	—	—	—
104	—	—	—	—
105	—	—	—	—
106	—	—	—	—
107	—	—	—	—
108	—	—	—	—
109	—	—	—	—
110	—	—	—	—
111	—	—	—	—
112	—	—	—	—
113	—	—	—	—
114	—	—	—	—
115	—	—	—	—
116	—	—	—	—
117	—	—	—	—
118	—	—	—	—
119	—	—	—	—
120	—	—	—	—
121	—	—	—	—
122	—	—	—	—
123	—	—	—	—
124	—	—	—	—
125	—	—	—	—
126	—	—	—	—
127	—	—	—	—
128	—	—	—	—
129	—	—	—	—
130	—	—	—	—
131	—	—	—	—
132	—	—	—	—
133	—	—	—	—
134	—	—	—	—
135	—	—	—	—
136	—	—	—	—
137	—	—	—	—
138	—	—	—	—
139	—	—	—	—
140	—	—	—	—
141	—	—	—	—
142	—	—	—	—
143	—	—	—	—
144	—	—	—	—
145	—	—	—	—
146	—	—	—	—
147	—	—	—	—
148	—	—	—	—
149	—	—	—	—
150	—	—	—	—
151	—	—	—	—
152	—	—	—	—
153	—	—	—	—
154	—	—	—	—
155	—	—	—	—
156	—	—	—	—
157	—	—	—	—
158	—	—	—	—
159	—	—	—	—
160	—	—	—	—
161	—	—	—	—
162	—	—	—	—
163	—	—	—	—
164	—	—	—	—
165	—	—	—	—
166	—	—	—	—
167	—	—	—	—
168	—	—	—	—
169	—	—	—	—
170	—	—	—	—
171	—	—	—	—
172	—	—	—	—
173	—	—	—	—
174	—	—	—	—
175	—	—	—	—
176	—	—	—	—
177	—	—	—	—
178	—	—	—	—
179	—	—	—	—
180	—	—	—	—
181	—	—	—	—
182	—	—	—	—
183	—	—	—	—
184	—	—	—	—
185	—	—	—	—
186	—	—	—	—
187	—	—	—	—
188	—	—	—	—
189	—	—	—	—
190	—	—	—	—
191	—	—	—	—
192	—	—	—	—
193	—	—	—	—
194	—	—	—	—
195	—	—	—	—
196	—	—	—	—
197	—	—	—	—
198	—	—	—	—
199	—	—	—	—
200	—	—	—	—
201	—	—	—	—
202	—	—	—	—
203	—	—	—	—
204	—	—	—	—
205	—	—	—	—
206	—	—	—	—
207	—	—	—	—
208	—	—	—	—
209	—	—	—	—
210	—	—	—	—
211	—	—	—	—
212	—	—	—	—
213	—	—	—	—
214	—	—	—	—
215	—	—	—	—
216	—	—	—	—
217	—	—	—	—
218	—	—	—	—
219	—	—	—	—
220	—	—	—	—
221	—	—	—	—
222	—	—	—	—
223	—	—	—	—
224	—	—	—	—
225	—	—	—	—
226	—	—	—	—
227	—	—	—	—
228	—	—	—	—
229	—	—	—	—
230	—	—	—	—
231	—	—	—	—
232	—	—	—	—
233	—	—	—	—
234	—	—	—	—
235	—	—	—	—
236	—	—	—	—
237	—	—	—	—
238	—	—	—	—
239	—	—	—	—
240	—	—	—	—
241	—	—	—	—
242	—	—	—	—
243	—	—	—	—
244	—	—	—	—
245	—	—	—	—
246	—	—	—	—
247	—	—	—	—
248	—	—	—	—
249	—	—	—	—
250	—	—	—	—
251	—	—	—	—
252	—	—	—	—
253	—	—	—	—
254	—	—	—	—
255	—	—	—	—
256	—	—	—	—
257	—	—	—	—
258	—	—	—	—
259	—	—	—	—
260	—	—	—	—
261	—	—	—	—
262	—	—	—	—</td

Table VI. Nicotine Levels in Food Courts

sample	smokers no.	obd/ cigarettes no.	cigars or pipes, no.	closest smoker, ft	nicotine concn. μg/m³
37	6	6		15	1.6
38	16	16		4	1.6
39	8	11		4	2.1
40	7	7	1	7	2.5
42	17	19		15	3.0
41	34	34		6	3.1

correlation between the nicotine concentration and the above-mentioned factors) for the regressions are 0.0001, 0.0001, 0.776, and 0.396, respectively. A relationship is thus indicated between nicotine concentration and both the number of cigarettes and number of smokers. If sample 36 is eliminated from the regression analysis for nearest smoker, then  $p = 0.03$ , indicating a correlation between nicotine concentration and this parameter. Attempts to increase the degree of correlation by normalizing for various combinations of these factors were unsuccessful. No data were acquired regarding other factors that could have some impact on the nicotine concentration, such as the history of the number of cigarettes smoked prior to sampling, and the direction of air flow in the restaurants.

In Table VI are listed the data from samples acquired in the food courts of the shopping malls. The range of nicotine concentrations from the mall food court samples was  $1.6\text{--}3.1 \mu\text{g}/\text{m}^3$  with an arithmetic mean and standard deviation of  $2.3 \pm 0.7 \mu\text{g}/\text{m}^3$ . Although use of the geometric mean could not be justified for such a small sample set, it was calculated for comparison purposes and is the same as the arithmetic value. The average nicotine concentration for these samples is lower than that from the restaurant data (notwithstanding the large number of cigarettes observed to have been smoked) and is probably attributable to the much greater volumes of the food courts, which begin to approximate open-air restaurants.

### Conclusions

A procedure and experimental arrangement for the determination of personal exposure to concentrations of nicotine in indoor environments has been developed that has a low detection limit and is unobtrusive in its use. Developmental studies have again pointed to the need for the use of a basic compound for sample modification or desorption enhancement when trace quantities of nicotine are being processed or analyzed. The Tenax method has been applied to the determination of nicotine concentrations in a number of restaurants; results are comparable to those obtained by other researchers utilizing different methods for sampling in restaurants and other public places.

### Acknowledgments

We thank J. H. Moneyhun for his assistance in generation of simulated ETS atmospheres in the stainless steel chambers and in sample acquisition. We also thank C. D. Varnadore for his assistance in sample acquisition.

Registry No. TEA, 121-44-8; Tenax GC, 24938-68-9; nicotine, 54-11-5.

### Literature Cited

- (1) Brain, J. D.; Barry, B. E. In *Indoor Air and Human Health: Proceedings of the Seventh Life Sciences Symposium*; Gammie, R. B., Kaye, S. V., Eds.; Lewis Publishers, Inc.: Chelsea, MI, 1985; pp 215-225.
- (2) Badre, R.; Gullerm, R.; Abran, N.; Bourdin, M.; Dumas, C. Ann. Pharm. Fr. 1978, 36, 443-452.
- (3) Fischer, T.; Weber, A.; Grandjean, E. Int. Arch. Occup. Environ. Health 1978, 41, 267-280.
- (4) Weber, A.; Fischer, T. Int. Arch. Occup. Environ. Health 1980, 47, 209-221.
- (5) Hugod, C.; Hawkins, L. H.; Astrup, P. Int. Arch. Occup. Environ. Health 1980, 42, 21-29.
- (6) Repace, J. L.; Lowrey, A. H. Science (Washington, D.C.) 1980, 208, 464-472.
- (7) Spengler, J. D.; Treitman, R. D.; Tosteson, T. D.; Mage, D. T.; Soczek, M. L. Environ. Sci. Technol. 1985, 19, 700-707.
- (8) Repace, J. L.; Lowrey, A. H. Environ. Int. 1985, 11, 3-22.
- (9) Quant, F. R.; Nelson, P. A.; Sem, G. J. Environ. Int. 1985, 8, 223-227.
- (10) Hopkins, R.; Wood, L. E.; Sinclair, N. M. Clin. Pharmacol. Ther. 1984, 36(5), 788-795.
- (11) Hill, P. J.; Haley, N. J.; Wynder, E. L. J. Chronic Dis. 1983, 36(6), 439-449.
- (12) Matsuki, H.; Osaka, F.; Kasuga, N.; Sugita, M. Jpn. J. Public Health 1981, 28, 505-515.
- (13) Feyerabend, C.; Higenbottam, T.; Russell, M. A. H. Br. Med. J. 1982, 284, 1002-1004.
- (14) *Manual of Analytical Methods*; National Institute for Occupational Safety and Health: Cincinnati, OH, 1977; Part II, Vol. 3, Method No. S293.
- (15) Crouse, W. E.; Johnson, L. F.; Marmor, R. S. Beitr. Tabakforsch. Int. 1980, 10, 111-113.
- (16) Williams, D. C.; Whitaker, J. R.; Jennings, W. G. EHP. Environ. Health Perspect. 1985, 60, 405-410.
- (17) Green, C.; Ingebretsen, B.; Heavner, D. EHP. Environ. Health Perspect. 1985, 63, 249.
- (18) Eatough, D. J.; Benner, C.; Mooney, R. L.; Bartholomew, D.; Steiner, D. S.; Hansen, L. D.; Lamb, J. D.; Lewis, E. Eatough, N. L. Abstracts of Papers, 79th Annual Meeting of the Air Pollution Control Association, Minneapolis, MN, June 1986; Air Pollution Control Association: Pittsburgh, PA, 1986; paper 86-68.5.
- (19) Ogden, M. W.; Eudy, L. W.; Heavner, D. L.; Conrad, F. W.; Green, C. R. Presented at the 40th Tobacco Chemists' Research Conference, Knoxville, TN, October 1986.
- (20) Oldaker, G. B.; Conrad, F. C., Jr. Environ. Sci. Technol. 1987, 21, 994-999.
- (21) Oldaker, G. B.; Perfetti, P. F.; Conrad, F. C., Jr.; Conner, J. M.; McBride, R. L. Presented at the International Conference on Indoor Air Quality Tokyo, Japan, November 1987.
- (22) Hammond, S. K.; Leaderer, B. P.; Roche, A. C.; Schenker, M. Atmos. Environ. 1987, 21, 457-461.
- (23) Hammond, S. K.; Leaderer, B. P. Environ. Sci. Technol. 1987, 21, 497-500.
- (24) Proctor, C. Toxicol. Lett. 1987, 35, 131-134.
- (25) Muramatsu, M.; Umemura, S.; Okada, T.; Tomita, H. Environ. Res. 1984, 35, 218-227.
- (26) Muramatsu, M.; Umemura, S.; Fukui, J.; Arai, T. Int. Arch. Occup. Environ. Health 1987, 59, 545.
- (27) Gallant, R. F.; King, J. W.; Levine, P. L. Characterization of Sorbent Resins For Use in Environmental Sampling. U.S. Environmental Protection Agency, Office of Research and Development, Washington, DC, 1978; EPA-600/7-78-054, pp 15-18.
- (28) Sheldon, L. S.; Sparacino, C. M.; Pellizzari, E. D. In *Indoor Air and Human Health: Proceedings of the Seventh Life Sciences Symposium*; Gammie, R. B., Kaye, S. V., Eds.; Lewis Publishers, Inc.: Chelsea, MI, 1985; pp 335-349.
- (29) Jenkins, R. A.; Gayle, T. M.; Wike, J. S.; Manning, D. L. Sampling and Chemical Characterization of Concentrated Smoke, Toxic Materials in the Atmosphere. ASTM STP 786, American Society for Testing and Materials, 1982; pp 153-166.
- (30) Moneyhun, J. H.; Jenkins, R. A.; Guerin, M. R. Presented at the 40th Tobacco Chemists' Research Conference, Knoxville, TN, October 1986.

2023380388

- (31) Heavner, D. L.; Thome, F. A.; Eudy, L. W.; Green, C. A. Presented at the 39th Tobacco Chemists' Research Conference, Montreal, Quebec, Canada, October 1985.
- (32) Grubner, O.; First, M. W.; Huber, G. L. *Anal. Chem.* 1980, 52, 1755-1758.
- (33) Oldaker, G. B.; Conner, J. M.; Conrad, F. W., Jr.; McConnell, B. C.; Ogden, M. W.; Perfetti, P. F.; Stancill, M. W. Presented at the 41st Tobacco Chemists' Research Conference, Greensboro, NC, October 1987.
- (34) ACS Committee on Environmental Improvement and Subcommittee on Environmental Analytical Chemistry. *Anal. Chem.* 1980, 52, 2242.
- (35) Ogden, M. W., R. J. Reynolds Tobacco Co. Inc., Winston-Salem, NC, private communication, 1987.
- (36) Hinda, W. C.; First, M. W. *N. Engl. J. Med.* 1975, 292, 844-845.

Received for review May 16, 1988. Revised manuscript received October 31, 1988. Accepted November 11, 1988. Research sponsored by the Council for Tobacco Research Inc., under Interagency Agreement DOE No. ERD-85-471, CTR Project No. 132 with Martin Marietta Energy Systems, Inc., under Contract DE-AC05-84OR21400 with the U.S. Department of Energy.

## Aerobic and Anaerobic Microbial Dissolution of Toxic Metals from Coal Wastes: Mechanism of Action

Aroklesam J. Francis\* and Cleveland J. Dodge

Department of Applied Science, Brookhaven National Laboratory, Upton, New York 11973

Arthur W. Rose and Armando J. Ramirez

Department of Geosciences, The Pennsylvania State University, University Park, Pennsylvania 16802

Microbial dissolution of toxic metals from two types of coal-cleaning wastes, one high in pyrite and trace metals and low in organic carbon (fines fraction) and a second lower in trace metals and higher in organic carbon (filter cake), was studied. Under aerobic conditions, native autotrophic bacteria solubilized varying amounts of As, Cr, Cu, Mn, Ni, Pb, and Zn from the filter cake and fines fraction. Dissolution of the above metals was increased by severalfold when the inorganic nutrients N and P were supplemented. Under anaerobic conditions, concentrations of Fe, Cr, and Mn increased due to native anaerobic bacterial activity from filter cake amended with carbon and nitrogen. Concentrations of soluble Ni and Zn in filter cake decreased, probably due to sulfate reduction and formation of insoluble metal sulfides. Selective chemical extractions of coal wastes indicate that most trace metals were associated with pyrite, ferric oxides, and a soluble phase, possibly ferric sulfate. The predominant mechanism of dissolution of metals from coal wastes under aerobic conditions is due to bacterial oxidation of pyrite; under anaerobic conditions it is due to bacterial reduction of iron and manganese oxides and the release of trace metals coprecipitated with the oxides.

on dissolution of metals. In addition to autotrophic microbial activity, an increase in heterotrophic microbial activity due to biodegradation of organic compounds in the residue (8) also can have an appreciable effect on the dissolution, mobilization, and immobilization of toxic metals from the residues. However, the extent and mechanisms of metal dissolution from coal refuse under the oxidizing and reducing conditions commonly encountered in the field (9) are incompletely understood.

In this study, two samples of coal-cleaning residue, one high in trace metals and relatively low in organic carbon (fines fraction) and the second low in trace metals and relatively high in organic carbon (filter cake) were used to investigate the extent and mechanism of microbial dissolution of toxic metals by the native microflora in the residue under aerobic and anaerobic conditions.

### Materials and Methods

**Source of Samples.** Coal-cleaning residues (fines fraction and filter cake) were collected from active circuits of the coal-washing plant of the Bradford Coal Co., Bigler, Clearfield County, Pa, in July 1983. This plant processes a mixture of bituminous coals from central and northwestern Pennsylvania. The samples were collected in clean 5-gal polyethylene containers, sealed, and shipped to the laboratory in a cooler with ice. Upon receipt at the laboratory, the samples were immediately analyzed for microbiological and chemical characteristics. Unused portions of the samples were stored in air-tight containers in a refrigerator.

**Microbiological Analysis.** The total number of bacteria in the fines fraction and filter cake were enumerated by Acridine Orange direct counts (AODC) (10, 11). Total viable aerobic and anaerobic bacteria as colony-forming units (CFU) were determined by using Trypticase-soy agar (Difco) and 50% diluted thioglycolate medium (Difco), respectively (12). Sulfur- and iron-oxidizing bacteria were enumerated by the most probable number (MPN) technique. Iron-oxidizing bacteria were determined by using 9K medium (13). Sulfur-oxidizing bacteria were determined by using thiosulfate medium containing the following:  $(\text{NH}_4)_2\text{SO}_4$ , 1.3 g; K<sub>2</sub>HPO<sub>4</sub>, 0.28 g; MgSO<sub>4</sub>·7H<sub>2</sub>O, 0.25 g; CaCl<sub>2</sub>·2H<sub>2</sub>O, 0.07 g; and 900 mL of distilled water.

### Introduction

Over 3 billion tons of coal-cleaning residues have accumulated in the United States and the current levels of production exceed 100 million tons per year. Nearly one-third of the mined coal is discarded after physical cleaning. This refuse varies in size and generally contains waste coal, slate, carbonaceous and pyritic shales, clay, and other impurities associated with a coal seam (1). Currently, most coal-preparation plants dewater the fine refuse and dispose of it, along with coarse refuse, in landfills or disposal ponds. The types of contaminants released from the disposal areas include organic compounds, metal ions, and acidity primarily due to chemical and microbiological action.

Bacterial oxidation of pyrite and metal sulfide minerals by *Thiobacillus ferrooxidans* and *Thiobacillus thiocyanococcus* has been extensively studied (2, 3). Although a variety of other types of microorganisms have been found in coal waste (4-7), there is limited information on their effects

2023380389

## CORRESPONDENCE

1105

2. Jacob P III, Wilson M, Benowitz NL. Improved gas chromatographic method for the determination of nicotine and cotinine in biologic fluids. *J Chromatogr* 1981; 223:61-70.
3. Russell MAH, Wilson G, Patel UA, Feyereisen C, Cole PV. Plasma nicotine levels after smoking cigarettes with high, medium, and low nicotine yields. *Br Med J* 1975; 2:414-6.
4. Benowitz NL, Jacob P III, Jones RT, Rosenberg J. Interindividual variability in the metabolism and cardiovascular effects of nicotine in man. *J Pharmacol Exp Ther* 1982; 231:368-73.

## COST OF SMOKING

To the Editor: Increasingly, the public and physicians are concerned about smoking as the major preventable cause of illness and death in our society. In addition, in this era of cost consciousness in medicine it is important to recognize the economics of the cigarette-smoking habit. Massachusetts had a population of 5,737,037 according to the 1980 census.<sup>1</sup> The American Cancer Society calculates that 25 per cent of the entire population smokes—or 1,434,259 Massachusetts citizens. The Commonwealth of Massachusetts imposes a 21-cent-per-package tax on cigarettes, and in 1980 collected \$1,444,018,195 by means of this levy,<sup>2</sup> indicating that about 685,801,000 packages of cigarettes were sold, or 1.3 packages per smoker per day, or 478 packages per year. The Office of State Health Planning estimated that in Massachusetts in 1980 total medical costs directly related to smoking were \$7,531,907,000 or \$1,313 per capita (Chenotakis A, unpublished data). This was believed to be a conservative figure, since other estimates for 1980 were as high as \$8,500,329,000, or \$1,478 per capita.

Studies have indicated that almost 10 per cent of all medical costs are directly related to tobacco smoking.

Luce and Schweizer<sup>3</sup> in 1976 estimated that the direct medical costs of smoking were 7.8 per cent of all medical costs but acknowledged that "our estimates are . . . understated." They noted a "smoking factor" of 20 per cent in all neoplasms at that time, but taking into account the continued rapid escalation of lung cancer in both men and women, currently 30 per cent of all cancer deaths in the United States are related to smoking,<sup>4</sup> which is but one example of the increase in medical costs related to tobacco.

Thus, non-smoking-related per capita medical costs in 1980 are estimated at \$1,182 (90 per cent × \$7,531,907,000 ÷ 5,737,037), or \$131 (10 per cent) less than the \$1,313 per capita figure for smoking-related medical costs. Annual medical costs among smokers are \$1,701 (\$1,182 + [8753,190,700 ÷ 1,434,259]), or \$525 per smoker per year extra. Most striking of all is the realization that the additional \$525 in medical costs per year per smoker is the equivalent of \$1.10 per package of 20 cigarettes (\$525 ÷ 478 packs), or over 5 cents per cigarette!

There are a variety of suggestions on how to shift this enormous financial burden from the nonsmokers to the smokers who incur this risk, and perhaps some innovative approaches in taxation or insurance should be considered. Besides the Surgeon General's warning about health risks printed on cigarette packages, it would be informative for the smokers to recognize that other people are paying even more than the cost of the package of cigarettes to subsidize the consequences of their habit.

BLAKE CADY, M.D.  
Boston, MA 02213      New England Deaconess Hospital

1. 1980 United States census. Washington, D.C.: United States Bureau of the Census, 1981.
2. Tobacco Estate Bureau: Cigarette Estate of Massachusetts. Massachusetts Department of Revenue, 1980.
3. Luce BR, Schweizer SO. Smoking and alcohol abuse—a comparison of their economic consequences. *N Engl J Med* 1978; 298:569-71.
4. American Cancer Society. *Cancer facts and figures*. New York: American Cancer Society, 1982.

## MORE ON BIMANUAL DEXTERITY IN BASEBALL PLAYERS

To the Editor: Although McLean and Chaitzak,<sup>1</sup> the authors of the letter analyzing bimanual dexterity in major-league baseball play-

## PASSIVE ABSORPTION OF NICOTINE IN AIRLINE FLIGHT ATTENDANTS

To the Editor: There is concern that nonsmokers may suffer adverse health effects from exposure to side-stream cigarette smoke (March 27, 1980, issue).<sup>1</sup> Airline flight attendants are regularly exposed to cabin air that is contaminated with cigarette smoke. We conducted a study to determine how much carbon monoxide and nicotine are absorbed by nonsmoking flight attendants during transoceanic commercial flights.

Participants were sought among nonsmoking flight attendants on the San Francisco-Tokyo-San Francisco route. Before the departure of the flight, blood samples were obtained from participants, and they completed questionnaires and were given containers for a urine collection during the return leg of the flight (Tokyo to San Francisco). Within one hour after their return to San Francisco, a second blood sample was obtained.

Six nonsmoking women between 30 and 40 years of age participated in the study. Only one participant lived with someone who smoked. All were full-time flight attendants who worked 68 to 73 hours per month. Five of the six attendants served in smoking sections on the Tokyo-to-San Francisco leg of the flight. The blood carboxyhemoglobin concentration (mean ± S.D.) was 1.0±0.2 per cent before takeoff, and there was no marked difference in the post-flight sample (0.7±0.2 per cent). Blood nicotine concentrations, measured by gas chromatography,<sup>2</sup> increased in five of six attendants, from a mean of 1.6±0.8 µg per milliliter (range, 0.8 to 2.7) to 3.2±1.0 µg per milliliter (range, 1.6 to 4.5;  $P < 0.05$  by Student's paired t-test). These concentrations are extremely low compared with concentrations (15 to 45 µg per milliliter) found in typical cigarette smokers.<sup>3</sup> Urinary excretion of nicotine during the eighteen-hour flight averaged 12.9±6.5 µg (range, 6.8 to 21.7) and was lowest in the flight attendant who worked in the nonsmoking section. On the basis of urinary-excretion data and known pharmacokinetic data for nicotine,<sup>4</sup> we estimated that the flight attendants, on the average, were exposed to 0.12 to 0.25 mg of nicotine, and that the flight attendant exposed to the largest amount of smoke received 0.22 to 0.43 mg during the flight. We conclude that there is passive absorption of nicotine from tobacco smoke by flight attendants during a transoceanic flight but that the quantity consumed (equivalent to one cigarette) is relatively small compared with that consumed by cigarette smokers, and the concentrations achieved are unlikely to have physiologic effects.

DONNA FOLLAST, M.D.  
NEAL L. BENOWITZ, M.D.  
CHARLES E. BECKER, M.D.  
San Francisco General Hospital  
Medical Center

San Francisco, CA 94110

1. Loeffelholz C, Liu BM. (Passive) nicotine versus (voluntary) nicotine. *N Engl J Med* 1980; 302:742-3.

2023380390

## Estimation of Effect of Environmental Tobacco Smoke on Air Quality within Passenger Cabins of Commercial Aircraft

Guy B. Oldaker III\* and Fred C. Conrad, Jr.

Research and Development Department, Bowman Gray Technical Center, R. J. Reynolds Tobacco Company, Winston-Salem, North Carolina 27102

Nicotine was measured in passenger cabins of Boeing B727-200, B737-200, and B737-300 aircraft in order to estimate the levels of environmental tobacco smoke (ETS) and to assess the effectiveness of smoker segregation as a means of reducing nonsmokers' exposure to ETS. Integrated sampling was performed at seats in smoking and no-smoking sections on flights averaging 55 min. Nicotine was collected on XAD-4 resin and analyzed by gas chromatography with nitrogen-phosphorus detection. Results indicate that significant nicotine concentration gradients exist in cabins and that concentrations increase in magnitude from no-smoking sections to smoking sections. The mean nicotine concentration for samples acquired in no-smoking sections was  $5.5 \mu\text{g}/\text{m}^3$ ; in smoking sections of aircraft the mean nicotine concentration was  $9.2 \mu\text{g}/\text{m}^3$ . These concentrations correspond to estimated mean exposures of 0.0041 and 0.0082 cigarette equivalent per flight, respectively.

### Introduction

In the U.S., commercial airlines are required during flights to segregate smokers in order to reduce the exposure of nonsmokers to environmental tobacco smoke (ETS), defined as the mixture of diluted and aged sidestream smoke and exhaled mainstream smoke. Since the implementation of this requirement (1), its consequences for cabin air quality have not been systematically studied. Although data relative to the levels of ETS in aircraft are contained in a report (2) issued jointly by the U.S. Department of Health, Education and Welfare (DHEW) and the U.S. Department of Transportation (DOT), they were obtained before segregation was required.

The literature contains only one report dealing with the quantitation of ETS levels in passenger cabins. Muramatsu et al. (3) reported the results of seven samples of vapor-phase nicotine collected during Japanese domestic flights. These researchers, however, provided no information on sampling locations. The choice of nicotine as an indicator of ETS reflects the fact that this compound is uniquely specific for tobacco smoke. At the time these results were reported, the relation between vapor-phase nicotine and ETS had not been characterized. Eudy et al. (4) have since then shown that at least 95% of the nicotine associated with ETS exists in the vapor phase.

For the study reported here, vapor-phase nicotine was sampled in passenger cabins of U.S. domestic aircraft in order to gain additional information regarding ETS levels therein and to assess the effectiveness of smoker segregation as a means of reducing the exposure to ETS by persons seated in no-smoking sections. Samples were collected unobtrusively with systems contained in ordinary briefcases in order not to disturb the behavior of passengers or to disrupt airline operations.

### Experimental Section

**Sampling System.** Samples were acquired with sampling systems contained in briefcases that were carefully designed to be inconspicuous (Figure 1). Brass sample

inlet and exhaust ports and the on-off switch were located on the front of each briefcase and were positioned symmetrically about the handle. Sample ports were fashioned from 0.25-in. o.d. Swagelok port connectors. Tubing extensions of port connectors were removed, and the resulting flat surfaces were polished. In addition, one of the port connectors was drilled out to a diameter of 0.25 in. to accommodate 6-mm o.d. XAD-4 sorbent tubes.

Major components of the system for sampling nicotine included an XAD-4 sorbent tube and a constant-flow sampling pump (both obtained from SKC, Inc., Eighty Four, PA). Each XAD-4 sorbent tube was positioned through the fitting on the briefcase's front so that approximately 3 mm of the tube's tip projected. Sorbent tube outlets were connected to sampling pumps with short lengths of rubber tubing. Sampling pumps were calibrated with a film flow meter, and flow rates were set at 1 L/min. Calibrations were confirmed with a mercury film flow meter. Flow rates were computed at standard conditions: 298 K (25 °C) and 760 Torr. Temperature and pressure data for adjusting calibration results to standard conditions were obtained from a mercury-in-glass thermometer and a mercury-in-glass barometer, respectively. According to protocol, calibrations were checked at weekly intervals throughout the study. Results from sampling were judged acceptable if the calibrations remained with  $\pm 5\%$ .

**Sampling Procedure.** A written sampling protocol was prepared in conjunction with the study. Persons conducting the sampling were provided with this protocol and also were orally briefed at the start of the study. In addition, persons conducting the sampling had security clearances that permitted them to pass through security stations without revealing the briefcases' contents. All but 14 of the samples were acquired by airline employees, who agreed to participate in the study gratis. The protocol directed that none of the persons conducting the sampling was to smoke during the times when samples were acquired. All sampling operations were performed during scheduled commercial flights that involved business unrelated to the study. Persons conducting the sampling selected flights strictly on the basis of availability and made no effort to select among aircraft types. None of the aircraft that figured in the study had first-class compartments; each aircraft had one smoking section and one no-smoking section.

Sampling was performed during the times when carry-on items such as briefcases could be unobtrusively removed from beneath seats. These times correspond to the times when smoking is permitted in the passenger cabins. Owing to the airline company's seating policy, most samples were obtained at boundary regions between smoking and no-smoking sections. Boundary regions included the last two rows in no-smoking sections adjacent to smoking sections.

Positioning of briefcases during the sampling depended on whether unoccupied seats were available. According to protocol, if an empty seat existed next to the person conducting the sampling, the briefcase was placed in the empty seat and oriented vertically; otherwise, the briefcase was placed in a horizontal position on the sampler's lap.

2023380391

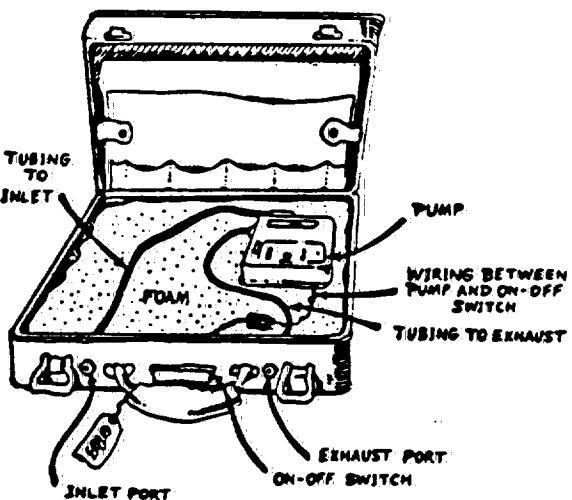


Figure 1. Briefcase sampling system.

with the sampling ports directed away from the body. When briefcases were oriented vertically, samples were acquired within approximately 15 cm of an adult passenger's breathing zone; when briefcases were oriented horizontally, this distance was approximately 45 cm. Airflow to sampling ports was unobstructed. In addition, the protocol specified that the air vents (gaspers) located in the passenger service unit above seats occupied by the briefcase samplers were to be closed during the sample acquisition. The protocol specified that samples be placed in a freezer within 24 h of acquisition.

Barometric pressure was measured on four flights. For the first of these, a hand-held altimeter calibrated against a mercury-in-glass barometer was employed. Response was approximated with a linear least-squares numerical method ( $R^2 = 0.999$ ). Use of the altimeter was determined to be overly conspicuous and burdensome, and consequently its use was discontinued. Additional pressure data were provided by a pressure transducer (Omega Engineering, Inc., Stamford, CT) installed in the briefcase. The transducer was calibrated with a mercury-in-glass barometer and interfaced with a 21X Micrologger (Campbell Scientific, Inc., Logan, UT).

**Analytical Procedure.** Two methods were used to analyze nicotine, both representing enhancements of the method (5) developed by the National Institute of Occupational Safety and Health (NIOSH). From the beginning of the study to 14 January 1986 (corresponding to sample number 36), samples were analyzed with a Model 5880A gas chromatograph equipped with a nitrogen-phosphorus detector (NPD) and a Model 7672A automatic sampler (Hewlett-Packard, Avondale, PA). The column used was a 30-m DB-WAX fused silica capillary with a 0.32-mm i.d. Injections were performed in splitless mode. Column temperature was programmed from 60 to 210 °C at 12 deg/min. Temperatures for the injector and detector were 250 and 300 °C, respectively. Quantitation was accomplished with the use of quinoline as an internal standard.

The method employed for the remainder of the study entailed the use of a 30-m DB5 megabore column with an internal diameter of 0.53 mm and a film thickness of 1.5 µm. Temperatures for the injector and detector were 250 and 300 °C, respectively. Column temperature was programmed from 150 to 175 °C at 5 deg/min. In addition, the ethyl acetate solvent was modified to contain 0.01% (v/v) triethylamine.

Chromatographic systems were calibrated at a minimum of five concentration levels for each set of analyses.

Reagent-grade nicotine for these standards was obtained from Eastman Kodak and was used as received. This reagent was stored in a freezer. Field samples were analyzed once; calibration standards were analyzed in duplicate before and after field samples. Results for calibration standards were used in conjunction with a linear least-squares program to compute nicotine levels of field samples and blanks. At least two sample blanks were analyzed with each set of field samples. Nicotine desorption efficiency from XAD-4 resin was determined according to the NIOSH procedure to be 0.92.

Exposures were estimated by computing "cigarette equivalents" from nicotine concentration results and associated sampling times. A breathing rate of 20 L/min (6), corresponding to light activity, was assumed for these calculations. Also assumed was a 1983 sales-weighted average cigarette delivering 0.93 mg of nicotine (7) as measured by the Federal Trade Commission (FTC) method (8, 9).

### Results and Discussion

Results of measurements performed in no-smoking and smoking sections of B727-200, B737-200, and B737-300 aircraft are shown in Tables I and II, respectively. These Boeing aircraft types differ among themselves in terms of seating capacity, location of boundary between smoking and no-smoking sections, and operation and design of heating, ventilating, and air conditioning (HVAC) systems. Ventilation systems of B727-200 and B737-200 aircraft are "once-through" systems; i.e., they are incapable of recirculating air within the cabins. Ventilation systems of B737-300 aircraft, on the other hand, recirculate approximately 40% of the air in the passenger cabins (10). Recirculated air is passed through a prefilter and then through a hospital-grade filter (95% efficient for 0.3-µm particles) to remove particulate matter. The population of aircraft studied may be considered representative inasmuch as modern commercial aircraft utilize both ventilation conditions and the three Boeing aircraft types constitute approximately 50% of the U.S. domestic, commercial aircraft fleet (11).

Seat entries in the tables identify sampling locations. Numbers indicate seating rows, which are numbered from front to back of the aircraft. Accompanying letters designate positions in rows, which for all rows sampled contained six seats, three seats on each side of the aisle. For the aircraft studied, the location of the smoking boundary is variable, depending for each flight on aircraft type, flight demographics, and number of passengers requesting to sit in either of the sections.

The tabulated number of passengers seated in the smoking sections provides an upper estimate of the number of smokers on a particular flight and allows estimation of an upper limit for the number of cigarettes smoked. The number of cigarettes smoked during a flight was estimated by assuming a smoking rate of two cigarettes per hour per passenger seated in the smoking section. This rate is one of two contained in the joint DHEW/DOT report (2); the other measured rate is 0.9 cigarette per smoking passenger per hour. Halfpenny and Starrett (12), providing the only other estimate, report 1.34 cigarettes per smoking passenger per hour.

The number of passengers seated in the smoking section was quantified for only a portion of the study. This aspect of the data acquisition process reflected the fact that the study was implemented in phases in order to ensure the quality of results; thus, each successive phase of the study was implemented when data-reliability objectives were met.

2023380392

Table I. Results from Samples Collected in No-Smoking Sections of B727-200, B737-200, and B737-300 Aircraft

sample	aircraft type	seat	no. in smoking section	no. of cig smoked (estd)	sampling time, min	nicotine		cig equiv
						µg	µg/m³	
85	737-200	1F*	15	20	41	ND (0.02)	ND (0.5)	NA
1	727-200	3D*	20	49	73	ND (0.02)	ND (0.03)*	NA
40	737-200	16C	20	30	45	ND (0.02)	ND (0.04)	NA
64	737-300	16E	5	8	50	ND (0.02)	ND (0.4)	NA
66	737-200	19B	25	76	65	ND (0.02)	ND (0.03)	NA
50	727-200	19B	12	26	85	0.04	0.6	0.0009
83	737-300	2B*	12	17	42	0.09	0.8	0.0007
41	737-200	12C	30	45	45	0.04	0.8	0.0008
26	737-300	15D	NA	NA	50	0.10	1.5	0.0016
82	737-200	11E	NA	NA	66	0.24	1.6	0.0022
69	737-200	9A*	25	63	76	0.17	1.7	0.0027
46	737-200	15C	1	1	41	0.06	1.8	0.0016
3	727-200	19F	20	49	73	0.14	1.9*	0.0029
9	737-300	4F*	NA	NA	39	0.10	2.1	0.0018
2	727-200	19E	20	49	73	0.17	2.3*	0.0037
72	737-200	NA	25	44	53	0.15	2.3	0.0027
32	727-200	19C	NA	NA	40	0.11	2.4	0.0021
75	737-300	6B*	NA	NA	32	0.11	2.7	0.0018
7	727-200	22C	NA	NA	132	0.44	2.7	0.0077
21	737-200	14D	NA	NA	51	0.27	3.3	0.0036
44	727-200	20C	14	16	34	0.13	3.4	0.0025
39	737-300	19C	7	13	57	0.28	4.3	0.0052
77	737-200	12B*	6	7	36	0.21	4.4	0.0034
31	737-200	15E	NA	NA	30	0.20	6.4	0.0041
33	737-200	15D	NA	NA	45	0.33	6.4	0.0062
17	737-200	15D	NA	NA	42	0.39	6.8	0.0062
20	737-200	15C	NA	NA	75	0.88	7.2	0.0117
10	737-300	11D*	NA	NA	39	0.40	8.1	0.0068
29	737-200	15D	20	30	45	0.47	10.0	0.0097
13	737-200	11C*	NA	NA	20	0.27	10.1	0.0044
19	737-200	15D	NA	NA	20	0.27	10.1	0.0044
38	737-200	15C	7	12	50	0.64	11.2	0.0120
81	737-300	15E	20	11	17	0.45	11.7	0.0043
80	737-200	15A	15	56	111	3.76	12.8	0.0306
71	737-200	15B	6	8	41	0.71	14.3	0.0126
70	727-200	20E	40	88	66	1.36	14.6	0.0207
30	737-200	15B	25	29	35	0.53	14.6	0.0110
54	737-200	16B	8	15	55	0.89	16.4	0.0182
73	737-200	15C	30	48	48	0.95	16.6	0.0172
58	737-300	16E	10	15	45	0.80	16.7	0.0162
16	737-300	16A	NA	NA	37	1.03	17.2	0.0137
53	737-200	15C	5	8	45	0.95	17.9	0.0173
42	737-200	15C	16	30	56	1.26	19.5	0.0235
22	737-200	14D	NA	NA	50	1.74	21.5	0.0231
57	737-200	15C	14	18	39	1.06	23.3	0.0196
61	727-200	20C	54	128	71	1.26	24.2	0.0369
15	737-200	15D	NA	NA	55	2.18	24.4	0.0288
74	737-200	15C	15	24	47	1.83	22.7	0.0330
18	737-200	15D	NA	NA	13	0.85	40.2	0.0112

\*Samples collected outside of boundary rows. \*Concentration at actual conditions.

and maintained. The number of active smokers on a particular flight, and thus the number of cigarettes smoked, was not quantified because this would have disrupted airline operations.

Nicotine results in each table are reported in the manner recommended by the American Chemical Society (13). For results below the limit of detection, ND signifies none detected, and the detection limit is shown in parentheses. For results below the limit of quantitation, the measured quantity is given, and the limit of quantitation is presented in parentheses.

Included in Tables I and II are the results of one experiment performed to assess the spatial variability of nicotine concentrations on a single flight. Four concurrent measurements were performed during a 73-min flight. One sample (sample 1) was acquired at seat 3D in the forward portion of the no-smoking section, two samples (samples 2 and 3) were acquired at adjacent seats 19E and 19F in the no-smoking section on the boundary with the smoking section, and one sample (sample 4) was obtained at seat

24F in the smoking section. The observed nicotine concentrations (at actual conditions) were <0.03, 2.3, 1.9, and 42.2 µg/m³, respectively. Twenty persons occupied the smoking section.

The results from this experiment show nicotine levels decreasing substantially from the smoking section to the no-smoking section. The smoker nearest seats 19E and 19F was seated one row distant on the opposite side of the aisle. The results suggest that nicotine (and therefore, ETS) concentration gradients may typically exist at boundary rows.

Bartlett's test for homogeneity of variances was used to test the nicotine concentration data. Test results supported a log-normal distribution. The concentration data were transformed to their logarithms in order to obtain homogeneity of variances and a normal distribution. The transformed data were then analyzed with a 3 × 2 factorial model ANOVA. Results indicate that the effect of aircraft type is not significant ( $P = 0.1802$ ). On the other hand, the results show the effect of seating section (either

Table II. Results from Samples Collected in Smoking Sections of B727-200, B737-200, and B737-300 Aircraft

sample	aircraft type	seat	no. in smoking section	no. of cig smoked (estd)	sampling time, min	nicotine		cig equiv
						µg	µg/m³	
35	737-200	16E	NA	NA	50	ND (0.004)	ND (0.08)	NA
68	737-200	15C	13	26	60	ND (0.02)	ND (0.03)	NA
87	737-200	17C	20	37	55	0.04 (0.08)	0.6 (1)	NA
65	737-300	19E	22	37	60	0.04 (0.08)	0.7 (2)	NA
45	727-200	20E	25	68	105	0.04	0.4	0.0009
59	727-200	20B	21	50	72	0.05	0.7	0.0010
27	737-200	16D	NA	NA	55	0.15	2.1	0.0024
60	737-200	15B	NA	NA	45	0.11	2.3	0.0022
49	737-200	14C	10	17	52	0.19	3.1	0.0035
6	727-200	22F	NA	NA	179	0.98	4.5	0.0172
83	737-200	30C	24	20	25	0.23	8.6	0.0046
24	737-300	17B	10	17	50	0.46	8.8	0.0095
48	727-200	19B	10	23	70	0.76	10.2	0.0164
28	727-200	21B	17	32	57	0.62	10.5	0.0129
14	727-200	19D	NA	NA	60	1.07	11.0	0.0142
6	727-200	22B	NA	NA	91	1.86	14.9	0.0291
47	737-300	16C	35	123	105	2.05	18.7	0.0423
56	737-200	15C	11	6	16	0.42	22.1	0.0076
51	737-200	18E	7	11	45	1.44	30.2	0.0293
76	737-300	20E	15	19	37	2.01	39.5	0.0314
4	727-200	24F	20	48	72	3.07	42.2*	0.0653
78	737-300	23F	22	30	41	4.82	45.0	0.0397
62	737-200	17D	20	17	25	1.51	57.1	0.0307
79	737-300	22D	22	84	114	16.79	59.8	0.1466
52	737-300	16B	23	38	60	4.06	76.7	0.0825
43	737-200	18C	23	31	40	6.18	112.4	0.0867

\*Concentration at actual conditions.

smoking or no smoking) to be significant ( $P = 0.0477$ ) as well as the effect of interaction, namely, aircraft  $\times$  seating section ( $P = 0.0766$ ). The model analyzed interaction with a type III sum of squares, which compensates for an unbalanced number of data and any interaction effect on the main effect terms.

The data strongly suggest that the significance of the difference in the nicotine concentrations between smoking and no-smoking sections would have been greater if samples had been collected more evenly in no-smoking sections. Only 9 of the 48 samples associated with no-smoking sections were collected outside of the boundary region; these nine samples tend to be associated with lower nicotine concentrations. The significance of the aircraft-seating section interaction is expected in view of the fact that the areas of smoking and no-smoking sections and ventilation characteristics differ among the three aircraft types.

The number of persons seated in the smoking section and the sampling time, when employed as covariates for the  $3 \times 2$  factorial ANOVA model, were shown to be insignificant:  $P > 0.5437$  and  $P > 0.3221$ , respectively. The absence of significance for the former is exemplified by the  $0.4 \mu\text{g}/\text{m}^3$  result of sample 45 acquired in the smoking section of a B727-200 when occupied by 25 persons.

Table III summarizes the concentration results by aircraft type and seating section. Included in the table are data ranges and geometric means.

Mean nicotine levels in the aircraft investigated are substantially lower than mean levels observed in environments where the density of smokers is similar. For example, Muramatsu et al. (9) reported mean levels of 26.42, 38.73, and  $47.71 \mu\text{g}/\text{m}^3$  in student cafeterias, conference rooms, and automobiles, respectively. The design of the aircraft's HVAC systems accounts for both the observed relatively low nicotine concentration levels and the absence of significant correlation with number of smokers.

Figure 2 shows the patterns of air circulation along the cross-section of a B727-200 aircraft. (Diagrams for the

Table III. Summary of Results from Sampling Nicotine in Aircraft

aircraft type	seating section	N	nicotine concn., $\mu\text{g}/\text{m}^3$	
			range	mean
727-200	NS	10	ND (0.03)-24.2	2.6
	S	8	0.4-42.2	6.8
737-200	NS	29	ND (0.04)-40.2	7.7
	S	11	ND (0.08)-112.4	6.5
737-300	NS	10	ND (0.4)-17.2	4.2
	S	7	0.7 (2)-76.7	21.5
total	NS	49	ND (0.03)-40.2	5.5
	S	26	ND (0.08)-112.4	9.2

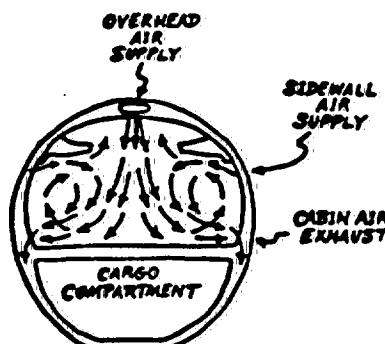


Figure 2. Schematic of airflow patterns for cross-section of B727-200 aircraft (17).

B737-200 and B737-300 aircraft are essentially the same.) Air supplies and exhausts are located in a manner that causes air to execute circular movements along a row of seats. Air enters the cabin from overhead vents and exits from vents located at foot level along cabin walls. Mirror-image circulation patterns distinguish port and starboard seats of each row. Air movement within a row also depends on the operation of overhead vents by passengers. Important aspects of the ventilation patterns shown in the

2023380394

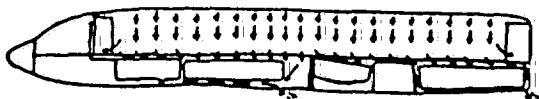


Figure 3. Schematic of airflow patterns along the length of B727-200 aircraft (17).

figures are that longitudinal movement of air in the cabins is suppressed, as is movement across the aisles. This longitudinal suppression of air movement is illustrated by Figure 3, showing ventilation patterns along the length of a B727-200's fuselage. (Diagrams for the B737-200 and B737-300 aircraft are essentially the same.) The high ventilation rates of the aircraft studied [for example, 26.5 air changes per hour for B727-200's, 22.7 air changes per hour for B737-200's, and 26.3 air changes per hour for B737-300's (10)] minimize the residence time of ETS in passenger cabins. Additionally, ETS will tend to remain within a single port or starboard row of seats owing to the effect of air movement patterns.

The effects of ventilation and air movement patterns and the relative isolation these effects impose on a port or starboard row of seats may account for those results where nicotine concentrations in smoking sections are below the limit of quantitation even though the sections are occupied by substantial numbers of passengers who presumably smoke. Similarly, the nicotine concentration results of this study, when viewed in light of the aircraft's ventilation characteristics, suggest that the port-starboard segregation approach utilized, for example, by European airlines, may be effective in reducing the exposure of persons seated in the no-smoking section to ETS.

The results of this study show that, to be adequate, models for air quality within aircraft cabins must account for the unique ventilation characteristics of aircraft. Models assuming the complete mixing of ETS in passenger cabins (14) are inappropriate for B727-200, B737-200, B737-300, and similar aircraft.

Most of the nicotine concentrations reported here have a high bias component due to the lack of barometric pressure data with which to adjust volumetric data from standard conditions to actual conditions. (Human respiration is not affected by the barometric pressures maintained in passenger cabins (15).) Barometric pressure data monitored on four sample runs indicate that concentration biases of up to 15% are possible.

The nicotine concentrations observed for this study are similar in magnitude to those reported by Muramatsu et al. (3). These workers, using a portable system attached to persons conducting the sampling, reported nicotine concentrations on Japanese domestic aircraft that ranged from 6.28 to 28.78  $\mu\text{g}/\text{m}^3$ . The mean concentration of the seven samples was 15.18  $\mu\text{g}/\text{m}^3$ . The authors concluded from these results that the exposure of persons to side-stream tobacco smoke, i.e., ETS, is very small. The authors, however, did not provide information regarding the types of aircraft, the sampling locations relative to the smoking sections, or the number of smokers; therefore, comparison with the results from the study reported here are limited.

Some researchers (3, 14, 16) have used the "cigarette equivalent" computational device to quantify exposure to ETS and thus to place such exposure in a convenient framework for discussion. Such computations assume an average daily breathing rate and an "equivalent cigarette" on the basis of the delivery of nicotine or "tar" in mainstream smoke. However, the term cigarette equivalent is inaccurate inasmuch as it suggests that persons thus ex-

posed are smoking, when in fact they are not. In addition, inhalation during smoking is deeper and more prolonged than during ordinary breathing, and breathing rates are variable rather than constant. Finally, the cigarette equivalent concept is highly manipulatable, because nicotine or tar deliveries vary over a wide range of values for different cigarette brands and because no single definition is currently recognized.

In spite of these shortcomings, the exposures represented by the nicotine levels observed for this study may perhaps be placed in perspective through use of the cigarette equivalent device. Accordingly, concentrations in no-smoking sections represent exposures ranging from 0.00004 to 0.037 cigarette equivalent per sampling period, with a geometric mean of 0.0041 cigarette equivalent per sampling period. Concentrations in smoking sections represent exposures ranging from 0.00008 to 0.15 cigarette equivalent per sampling period, with a geometric mean of 0.0082 cigarette equivalent per sampling period. These estimates in general indicate very low exposure relative to active smoking.

#### Conclusions

The results of this study show that (a) segregation significantly reduces the exposure of persons seated in no-smoking sections to ETS and (b) aircraft's HVAC systems are primarily responsible for effecting this reduction. In addition, the results indicate that average exposures to ETS are orders of magnitude less than exposures represented by smoking a single cigarette.

Additional research is needed in order to define more precisely and completely the effect ETS has on air quality in passenger cabins of commercial aircraft. Future studies should be expanded to include measurements of other ETS constituents and to involve wide-bodied aircraft on longer flights.

#### Acknowledgments

We thank B. C. McConnell and R. F. Walsh for their efforts in fabricating the briefcase sampling systems and M. W. Ogden, D. L. Heavner, L. W. Eudy, R. W. Hawley, B. J. Ingebrethsen, and C. R. Green for their technical assistance.

Registry No. Nicotine, 54-11-8.

#### Literature Cited

- (1) *Fed. Regist.* 1973, 38, 19146.
- (2) U.S. Department of Health, Education, and Welfare and U.S. Department of Transportation *Health Aspects of Smoking in Transport Aircraft*; U.S. Government Printing Office: Washington, DC, 1971.
- (3) Muramatsu, M.; Umemura, S.; Okada, T.; Tomita, H. *Environ. Res.* 1984, 35, 218.
- (4) Eudy, L. W.; Thome, F. A.; Heavner, D. L.; Green, C. R.; Ingebrethsen, B. J. Presented at the 39th Annual Tobacco Chemists' Research Conference, Montreal, Quebec, Canada, Oct 1985.
- (5) *Manual of Analytical Methods*; National Institute for Occupational Safety and Health: Cincinnati, OH, 1977; Part II, Vol. 3, Method No. S293.
- (6) International Commission on Radiological Protection Report of the Task Group on Reference Man; Pergamon: Oxford, 1974; p 346.
- (7) Anon. *Tob. Int.* 1983, 18(7), 68.
- (8) Pillsbury, H. C.; Bright, C. C.; O'Conner, K. J.; Irish, F. W. *J. Off. Anal. Chem.* 1969, 52, 458.
- (9) Wagner, J. R.; Thaggard, N. A. *J. Off. Anal. Chem.* 1979, 62, 229.
- (10) Yorozu, A. S., Boeing Commercial Airplane Co., Seattle, WA, personal communication, 1986.

- (11) "Air Transport 1985: The Annual Report of the U.S. Scheduled Airline Industry"; Air Transport Association of America: Washington, DC, June 1985.
- (12) Halfpenny, P. F.; Starrett, P. S. *ASHRAE J.* 1961, 9, 39.
- (13) ACS Committee on Environmental Improvement and Subcommittee on Environmental Analytical Chemistry. *Anal. Chem.* 1980, 52, 2242.
- (14) Repace, J. L.; Lowrey, A. H. *Science (Washington, D.C.)* 1980, 208, 464.
- (15) McFarland, R. A. *Human Factors in Air Transportation*; McGraw-Hill: New York, 1953; p 158.
- (16) Hinds, W. C.; First, M. W. *N. Engl. J. Med.* 1975, 222, 844.
- (17) "727-200 Maintenance Manual"; Boeing Commercial Airplane Co.: Seattle, WA; Boeing Document No. D6-45267.

Received for review June 23, 1986. Revised manuscript received March 18, 1987. Accepted June 20, 1987.

## Oligomerization of 4-Chloroaniline by Oxidoreductases

Kathleen E. Simmons, Robert D. Minard, and Jean-Marc Boileau\*

Department of Agronomy and Department of Chemistry, The Pennsylvania State University, University Park, Pennsylvania 16802

**■** Oxidation of aromatic amines by oxidoreductases can result in the formation of polycyclic aromatic products. We incubated 4-chloroaniline with horseradish peroxidase and with a laccase from the fungus *Trametes versicolor*. Qualitative and quantitative analyses were performed on the oligomeric products. Both enzymes generated eight oligomers, which were isolated and identified. On the basis of their structures and rates of formation, a reaction scheme for the oxidative oligomerization of 4-chloroaniline was proposed. The scheme shows that, once the substrate was enzymatically oxidized, free-radical coupling followed, and three dimeric intermediates were produced. Each of the dimers initiated a nonenzymatic reaction pathway, and the combined pathways accounted for the formation of the first eight stable 4-chloroaniline-derived oligomers.

### Introduction

Aniline-based herbicides readily decompose in the soil, but the resultant degradation products may be transformed into persistent xenobiotic species. Hydrolytic cleavage of the aliphatic portion of the herbicides produces substituted anilines, which often undergo oxidative polymerization and binding to soil organic matter. A study on the fate of substituted anilines in the soil found that at high concentrations (500 ppm) 40% of the applied material was recovered as polycyclic products and 50% was bound to soil organic matter (1). At low concentrations (1.25 ppm), 90% of an aniline soil residue was bound to soil constituents with only trace amounts recovered as extractable oligomers (2). It is likely that the processes leading to polymerization are also responsible for incorporation of the anilines into humic substances.

Models of oxidative reactions are essential for understanding the transformation of substituted anilines in soil. Numerous studies have been conducted on the one-electron oxidation of anilines using oxidoreductases, such as horseradish peroxidase and the laccases of *Trametes versicolor* and *Rhizoctonia praticola* (3-7). Some of the aniline-derived oligomers were structurally determined, but neither comprehensive product identifications nor quantitative analyses were reported.

In a previous work, we identified the structures of all products formed in the oxidoreductase-initiated polymerization of 4-chloroaniline and developed a method for substrate and product quantitation (8). In this investigation, we apply the quantitative method to follow 4-chloroaniline disappearance and product formations as a function of enzyme incubation times. We compare the product profiles resulting from the reactions catalyzed by horseradish peroxidase and the laccase of *T. versicolor*. On

the basis of product structures and their relative amounts, we postulate reaction pathways for the oxidative polymerization of substituted anilines in general and for 4-chloroaniline in particular.

### Materials and Methods

**Chemicals.** 4-Chloroaniline was purchased from Aldrich Chemical Co. (Milwaukee, WI) and was 98+ % pure as confirmed by thin-layer chromatography (TLC) and high-performance liquid chromatography (HPLC).

**Enzyme Assays.** Horseradish peroxidase with an RZ (Reinheitszahl) of 0.43 and an activity of 45 purpurogallin units/mg of solid was purchased from Sigma Chemical Co. (St. Louis, MO). A purpurogallin unit is defined as the amount of enzyme that forms 1.0 mg of purpurogallin from pyrogallol in 20 s at pH 6.0 and 20 °C. The absorbance change is measured at 420 nm.

The extracellular laccase of *T. versicolor* was isolated from growth media and purified as previously described (7). Laccase activity is given in DMP (2,6-dimethoxyphenol) units. A DMP unit is defined as the amount of enzyme causing a change in absorbance at 468 nm of 1.0 unit min<sup>-1</sup> at pH 4.2 of a 3.5-mL sample containing 3.24 μmol of 2,6-dimethoxyphenol. Absorbance was measured with a Model 2000 spectrophotometer (Bausch and Lomb, Inc., Rochester, NY).

Unless otherwise specified, enzyme assays were conducted in citrate-phosphate buffers (pH 4.2) with 1 μmol/mL 4-chloroaniline at 25 °C. Horseradish peroxidase assays contained 2.5 μmol/mL hydrogen peroxide and 0.012 purpurogallin unit/mL enzyme; 20 DMP units/mL was used in the laccase assays. Boiled enzymes served as controls.

**High-Performance Liquid Chromatography.** At the specified times (0-120 min), enzyme activity was halted by the addition of an equal volume of acetonitrile to a 2.5-mL aliquot of the assay solution. The 5.0-mL sample was then passed through a 0.2-μm pore Nylon 66 filter (Schleicher & Schuell, Keene, NH), and 175 μL was immediately analyzed by HPLC. All quantitative data points represent the average value of triplicate sample injections.

Analysis was performed on a Waters Associates (Milford, MA) high-performance liquid chromatograph. The system consisted of a U6K injector, M45 and 6000 pumps run by a Model 720 System Controller, a Lambda Max 450 LC spectrophotometer set at 280 nm (0.05 AUFS), and a Model 730 Data Module.

Reverse-phase separation was performed on a 15 cm × 4.6 mm Supelcosil LC18 (octadecylsilica) column of 5-μm particle size (Supelco, Inc., Bellefonte, PA). The mobile phase at a flow rate of 1.5 mL/min consisted of an aqueous

20238036

## Schlußwort-Zum Bereich des Spekulativen

Die gegen meinen Kommentar geschickte Leserzuschrift von F. Portheine hat sich substantiell mit den beiden zur Diskussion stehenden Veröffentlichungen und MMW-Interviews nicht auseinandergesetzt. Insbesondere aber hat F. Portheine keinen meines gegen die Aussagen von T. Hirayama vorgebrachten Kritikpunkte angesprochen, geschweige denn entkräften können. Im Bemühen, emotionslos und ausschließlich auf konkrete Argumente zu erwidern, bleiben zu seinen Ausführungen nur folgende Bemerkungen notwendig:

1. Tierexperimentell gewonnene Daten zum Problem des Passiv-Rauchens liegen im einschlägigen wissenschaftlichen Schriftum bislang nicht vor. Wenn nach F. Portheine trotzdem „die krebszeugende Wirkung des Passiv-Rauchens im Tierexperiment durch zahlreiche Untersuchungen einwandfrei gesichert“ ist, kann – da eine wesentliche Falschdarstellung nicht unterstellt wird – diese Auffassung nur aus einer Fehleinschätzung der Methodik durchgeführter Tierexperimente resultieren. Tatsächlich pflegen Versuchstiere kaum aktiv (= freiwillig) zu rauchen. Um die Gegebenheiten des Aktivrauchens beim Menschen zu simulieren, müssen daher die Versuchstiere zwangsläufig maschinell, d. h. passiv (= unfreiwillig) bestraucht werden. Daß diese Art des Zwaagsrauchens gelegentlich mit der Bezeichnung „Passivrauchen“ belegt wurde, muß im Hinblick auf die heute an diesen Begriff geknüpfte Problematik des „Mitrauchens“ als unglücklich empfunden werden. Dies berechtigt jedoch nicht, die im Tierexperiment so gewonnenen Erkenntnisse auf die Verhältnisse des Passivrauchens im heutigen Wortsinn (= Mitrauchen) zu übertragen.

2. Die von F. Portheine als Beweis für die Schädlichkeit des Passivrauchens angeführten Ergebnisse über den Gesundheitszustand von Kindern aktivrauchender Eltern

wurden spätestens durch die Untersuchungen von M. D. Lebowitz, D. B. Arner und R. Kundson (Int. Symp. on Indoor Air Pollution, Health and Energy Conservation, Amesbury/Mass., 13.-16. Oktober 1981) als Scheinkorrelation erkannt.

Unter dem Titel „The Effect of Passive Smoking on Pulmonary Function in Children“ gelangen die Autoren bei ihrer Studie zu der Endaussage: „It is apparent that household aggregation of pulmonary function, which is dependent on household aggregation of body mass, might affect the relationship of children's pulmonary function to paternal smoking. When these household aggregations are corrected for, there was no relationship of children's pulmonary function values to paternal smoking“. Hiermit wird einmal mehr deutlich, daß es gelegentlich durchaus hilfreich sein kann, nach der klinischen Plausibilität statistisch bestätigter Korrelationen zu fragen.

Bei der Zuschrift von H. Remmer befaßt sich ein erheblicher Teil des Beitrages mit dem aktiven Rauchen, dessen Schädlichkeit bei der vorgegebenen Thematik aber nicht zur Diskussion steht.

H. Remmer reduziert das kanzerogene Potential des Zigarettenrauchs auf das Vorkommen von Nitrosaminen, insbesondere von Dimethylnitrosamin (NDMA), wobei er gleichzeitig den Themenkomplex erheblich versimplifiziert. Tatsächlich haben sich N-Nitrosoverbindungen bis-

lang bei mehr als 20 Tierspezies als kanzerogen erwiesen. Mit guten Gründen kann für sie auch ein krebszeugendes Potential beim Menschen angenommen werden, wobei Aussagen zur Organotropie der kanzerogenen Wirkung allerdings vorerst nicht möglich sind (7). In dem hier zur Diskussion stehenden Zusammenhang muß festgestellt werden, daß H. Remmer passiv inhalierter Tabakrauch aber eben für die gesamte Nitrosaminbelastung des Menschen quantitativ wie möglicherweise auch qualitativ völlig falschen Stellenwert gibt. Nitrosamine kommen als flüchtige Verbindungen unabhängig von jedem Tabakabbrand offensichtlich ubiquitär vor, wobei in der Atemluft oft Konzentrationen erreicht werden, die weit über denjenigen liegen, die durch Tabakrauch erzeugt werden können. In Tabelle 1 finden sich einige Konzentrationsangaben für das von H. Remmer speziell angesprochene NDMA. Vergleichsweise sei in diesem Zusammenhang erwähnt, daß dem menschlichen Organismus mit der Nahrung täglich bis zu 1,1 µg NDMA zugeführt werden (9), und daß mit dem Hauptrstrom von 10 Filterzigaretten bis zu 0,06 µg NDMA aktiv inhalieren werden (3). In welchem Maße insbesondere mit der Nahrung aufgenommene nichtflüchtige Nitrosamine ein Gefährdungspotential darstellen, muß gegenwärtig noch offen bleiben, da die Analytik dieser Verbindungen heute noch nicht voll bereitstellt wird. Mit ziemlicher Sicher-

Tabelle 1: NDMA-Konzentrationen in der Luft und hieraus für eine Expositionzeit von 8 Stunden abgeschätzte inhalative NDMA-Aufnahme des Menschen

	NDMA-Wert	NDMA-Aufnahme in 8 h bei 10 Zigaretten
Offene Räume, in denen geraucht wird	0,02-0,05 µg/m³ (1)	0,096-0,24 µg
Innenräume von Autos	0,01-0,24 µg/m³ (2)	0,048-1,15 µg
Lederindustrie	0,07-0,83 µg/m³ (4)	0,336-3,98 µg
	bis zu 50 µg/m³ (6)	bis zu 240 µg

2023380397

heit darf aber bei allen Diskussionen um eine exogene Nitrosaminbelastung des Menschen die von H. Remmer nur andeutungsweise angesprochene In-vivo-Synthese von Nitrosaminen nicht unberücksichtigt bleiben. R. Tannenbaum (10) schätzt, daß beim Menschen so täglich bis zu 670 µg NDMA entstehen können. Mit diesem Kontext rücken die Überlegungen von H. Remmer zur gesundheitsschädigenden Relevanz des NDMA-Gehalts von Passivrauch eindeutig in den Bereich des Spekulativen.

Abschließend bleibt festzustellen, daß nur die wertfreie und vorurteilslose Diskussion den Raum schafft, in dem wissenschaftliches Suchen und Finden gedeihen kann. Jede emotionale Einstellung in der Argumentation kann der Wahrheitsfindung nur hinderlich sein. Dies gilt auch für die Fragen der gesundheitlichen Auswirkungen des Passivrauchens.

#### Literatur

1. Alumana, H.: Concentration of Dimethyl-nitrosamine in the Air of Smoke-Filled Rooms. persönliche Mitteilung 1981.
2. Brunnemann, K. D., Hoffmann, D.: Chemical Studies on Tobacco Smoke - LX. Analysis of Volatile Nitrosamines in Tobacco Smoke and Polluted Indoor Environment. IARC Scientific Publications No. 19 (1978) 243-256.
3. Brunnemann, K. D., Fink, W., Moser, F.: Oncology 37 (1980) 217-222.
4. Hammond, E. C., Selikoff, L. J.: Envir. Res. 24 (1981) 441-452.
5. Roundthaler, D. P., Reich, J., Fine, D. H.: Edin. Cosmet. Toxicol. 18 (1980) 147-151.
6. Selikoff, L. L., Hammond, E. C., Cruz, J.: J. Am. Med. Assoc. 204 (1968) 106-112.
7. Schmahl, D., Haber, M.: Oncology 37 (1980) 237-242.
8. Spiegelhalder, B.: Occupational Exposure to N-Nitrosamines, Vortrag anlässlich der Konferenz „Cancer Detection and Prevention“, London 1980.
9. Spiegelhalder, B., Eisenbrand, G., Preussmann, B.: Oncology 37 (1980) 211-216.
10. Tannenbaum, S. R.: Oncology 37 (1980) 222-225.

Prof. Dr. med. G. Lehner, Direktor des Zentralinstituts für Arbeitsmedizin, Adolph-Schünfelder-Str. 5, D-2000 Hamburg 76.

## „Humanes Sterben“: Wem hilft's tatsächlich?

Ein Jahr lang gibt es nun die Deutsche Gesellschaft für Humanes Sterben (DGHS). Sie umfaßt bei einer monatlichen Wachstumsrate von 80 bis 100 Mitgliedern nach eigenen Angaben über 1000 Menschen, die bereit sind, für einen jährlichen Mindestbeitrag von 50 DM human zu sterben. Das heißt auch, mit ausdrücklicher Billigung der passiven Sterbehilfe seitens der Medizin. Die DGHS sieht dabei die Herausgabe von Patientenverfügungen, die im Vollbesitz der psychischen und physischen Kräfte gemacht werden, als ihre bislang wichtigste Leistung an. Ebenso brachte sie eine Broschüre zuwege, die sich „Menschenwürdiges und selbstverantwortliches Sterben“ nennt und nur an Mitglieder ausgegeben wird, die mindestens seit einem Jahr dieser Organisation angehören. Auf diese Weise will sie – nach den Worten ihres Präsidenten Hans Hennine Aron – „Affekt-Taten ausschließen“. Und glaubt ernstlich, im Gegensatz zu anderen Interessenverbänden „internationaler Couleur, diejenige zu sein, welche „die größten Vorsichtsmaßnahmen trifft“.

Der DGHS-Vize Dr. Rasche, der die geheimnisvolle Schrift teils übersetzte, teils bearbeitete, teils neu konzipierte, wollte sie sogar nur an über 40jährige ausgehändigten wissen, weil – so weiß dieser „Landarzt mit großer Praxis“ (Rasche) – „erst in diesem Alter gewährleistet ist, daß der Mensch gefestigt ist, einen Beruf ausübt und sich Lebensvorstellungen gebildet hat“. Diese Einschränkung hatte er sich jedoch ohne Rücksprache mit den DGHS-Führungskollegen ausgedacht und wurde deswegen auch von seinem Präsidenten Aron entsprechend gerügt, der da meinte, es wäre wohl besser gewesen, das Thema zunächst intern zu regeln. Peinlicherweise fielen sowohl Rasches Lebensweisheiten als auch

Arons Distanzierung während der Pressekonferenz zur Ein-Jahres-Eloge.

So sehr die Aufgabenstellung der DGHS ist, so düstatisch erscheint ihre Bearbeitung oder gar Bewältigung. Zweifellos spricht alles – und gerade der medizinische Praktiker weiß oft ein garstig Lied davon zu singen – gegen ein langes Siechtum, möglicherweise unter erheblichen Schmerzen, in Intensivstationen, Klinikbetten oder zu Hause. Die Abnahme der ärztlichen Verantwortung durch die Patientenverfügungen gerade dann, wenn der Patient nicht mehr in der Lage ist, Entscheidungen zu treffen und zu äußern, bedeutet für den Therapeuten gewiß eine große Hilfe. Andererseits ist es fraglich, ob der Betroffene zur Zeit der Verfügungserklärung sich auch bewußt ist, was er tatsächlich verlangt oder erwarten kann.

Die DGHS sollte sich hier mit der Ausklärung und Geradestellung des Themas begnügen und nicht irgendwelche obskuren und esoterischen Broschüren auch noch zur Selbstbildung herausgeben. Daß sie das offensichtlich selbst auch so sieht, zeigt der deutliche Hinweis, sie mache sich mit der Schrift ja lediglich der Beihilfe zum Suizid schuldig. Und das ist natürlich, wie die Tat selbst, straffrei. Überdies, so betont die Gesellschaft, würde sie „bei einer gesetzlichen Regelung der aktiven Sterbehilfe diese Broschüre wieder zurückziehen“. Wem also soll sie tatsächlich helfen? Dem Gesetzgeber auf die Beine oder dem Mitglied unter die Erde?

Die Deutsche Gesellschaft für Humanes Sterben will ab 1982 eigene Ärztetage veranstalten.

Ingo Scholten, Merowingerstr. 22 a, D-4032 Gräfelfing.

CONCLUSION: IN THE REALM OF SPECULATION

by

G. Lehnert

Direktor des Zentralinstituts für Arbeitsmedizin  
Adolph-Schönfelder-Str. 5  
D-2000 Hamburg 76

The reader's letter from F. Portheine directed in opposition to my commentary did not substantially differ with the two publications and MMW-interviews under discussion. In particular, however, F. Portheine did not address any of my critiques brought forth against the statements of T. Hirayama, let alone refute them. In an effort to counter these without emotion and exclusively with concrete arguments, only the following remarks are required with respect to his statement:

1. Data obtained from animal experiments with respect to the problem of passive smoking are, to date, not available in the pertinent scientific literature. If, in accordance with F. Portheine, nevertheless "the carcinogenic effect of passive smoking is definitely substantiated through numerous investigations," - inasmuch as one can impute no conscious falsification - this conception can only result from an erroneous assessment of the methodology of the animal experiments carried out. It is indeed known, that experimental animals hardly smoke actively (= voluntarily). In order to simulate the circumstances of active smoking in humans, the experimental animals must be caused to smoke by force, i.e. passively (= involuntarily). The fact that this forced

2023380399

smoking is occasionally given the designation "passive smoking" must be seen as unfortunate in consideration of the problem of "co-smoking" currently associated with this concept. However, this is no justification for transferring the data thus obtained in animal experiments to the conditions of passive smoking in the present sense of the word (= co-smoking).

2. The results cited by F. Portheine as proof for the injuriousness of passive smoking with respect to the state of health of children of actively-smoking parents were recognized most recently as an apparent correlation by the investigations of M.D. Lebowitz, D. B. Armet and R. Kundson (Int. Symp. on Indoor Air Pollution, Health and Energy Conservation, Amherst, Mass., 13-16 October, 1981).

Under the title "The Effect of Passive Smoking on Pulmonary Function in Children", in their study, the authors arrived at the final statement "It is apparent that household aggregation of pulmonary function, which is dependent on household aggregation of body mass, might affect the relationship of children's pulmonary function to parental smoking. When these household aggregations were corrected for, there was no relationship of children's pulmonary function values to parental smoking." By virtue of this, it again becomes more clear that it is occasionally helpful to question the clinical plausibility of statistically substantiated correlations.

In the letter from H. Remmer, a considerable portion of the contribution is concerned with active smoking, the injuriousness of which is not under discussion with the given subject.

H. Remmer reduces the carcinogenic potential of cigarette smoke to the presence of nitrosamines, in particular of dimethylnitrosamine (NDMA) and in so doing, at the same time considerably oversimplifies the complexity of the subject. As a matter of fact, to date, N-nitroso-compounds have proven carcinogenic in more than 20 animal species. On good grounds, they can also be assumed to have a carcinogenic potential in humans, in which

2023380400

case statements on the organotropism of the carcinogenic effect, however, are certainly not possible [7]. In the relationship here under discussion it must be established that H. Remmer does not give a completely incorrect value for the overall nitrosamine burden of humans quantitatively or, possibly, also qualitatively. Nitrosamines obviously occur everywhere as volatile compounds independent of any type of tobacco combustion, in which case concentrations are often reached in respiratory air that are far above those that may be produced by tobacco smoke. Given in Table 1 are various data on concentrations for the NDMA particularly addressed by H. Remmer. By way of comparison, it might be mentioned in this connection that the human organism is daily supplied with up to 1.1 µg of NDMA with the nutrients [9] and that, in the mainstream of ten filter cigarettes, up to 0.06 µg of NDMA is actively inhaled [3]. The extent to which, in particular, non-volatile nitrosamines absorbed with the food represent a potential of endangerment must, for the present, remain an open question, for at present we have not yet mastered the analysis of these compounds. With some certainty, however, in all discussions of exogenous loading with nitrosamines in humans, the in-vivo synthesis of nitrosamines that is addressed by H. Remmer only in passing should not remain out of consideration. R. Tannenbaum [10] estimates, that in humans, in this manner up to 670 µg of NDMA may be produced daily. In this context, the considerations by H. Remmer with respect to the health-injurious relevance of the NDMA-content of passive smoking, clearly move into the range of speculation.

In conclusion, it remains to be stated that only impartial and unprejudiced discussion can create the space in which scientific research and

2023380401

discovery can thrive. Any emotional position in the argumentation can only be a hinderance to finding the truth. This applies also to the questions of the health effect of passive smoking.

Place	NDMA value	NDMA absorption in 8 hrs. at 10 l AMV
Rooms where smoking occurs	0.02-0.05 ug/m <sup>3</sup> [1] 0.01-0.24 ug/m <sup>3</sup> [2]	0.096-0.24 ug 0.048-1.15 ug
Car interiors	0.07-0.83 ug/m <sup>3</sup> [4]	0.336-3.98 ug
Leather industry	up to 50 ug/m <sup>3</sup> [6]	up to 240 ug

Table 1: NDMA concentrations in the air and hence the estimated inhaled NDMA absorption by humans during an exposure period of 8 hours

#### REFERENCES

- 1. Ahmann, H.: Concentration of Dimethyl-nitrosamine in the Air of Smoke-Filled Rooms. *personliche Mitteilung* 1981.
- 2. Brunnemann, K. D., Hoffmann, D.: Chemical Studies on Tobacco Smoke LX: Analysis of Volatile Nitrosamines in Tobacco Smoke and Polluted Indoor Environments. IARC Scientific Publication No. 14 (1976) 345-356
- 3. Brunnemann, K. D., Finlay, W., Moyer, F.: *Oncology* 37 (1981) 217-222
- 4. Hammond, E. C., Selikoff, I. J.: *Envir. Res.* 24 (1981) 141-152
- 5. Rounbehler, D. P., Reisch, J., Fine, D. H.: *Fd. Comité. Toxicol.* 18 (1980) 127-131.
- 6. Selikoff, I. J., Hammond, E. C., Churg, J.: *J. Am. Med. Assoc.* 252 (1984) 104-112.
- 7. Schmid, D., Habs, M.: *Oncology* 37 (1981) 237-242.
- 8. Spiegelhalder, B.: Occupational Exposure to N-Nitrosamines. *Vortrag anlaßlich der Konferenz „Cancer Detection and Prevention“ London 1981*
- 9. Spiegelhalder, B., Eisenbrand, G., Preussmann, R.: *Oncology* 37 (1981) 211-216
- 10. Tannenbaum, S. R.: *Oncology* 37 (1981) 233-235

2023380402

Oeff Gesundheitswes, 44(0):454-456,  
1982

ARE THERE REALLY IMPORTANT NEW FINDINGS ABOUT  
PASSIVE SMOKING ?

H. Schievelbein

Institute of Clinical Chemistry,  
The German Heart Center of Munich, State of Bavaria

Translated from German

2023380403

## ARE THERE REALLY IMPORTANT NEW FINDINGS ABOUT PASSIVE SMOKING?

By H. Schievelbein

Institute of Clinical Chemistry,  
the German Heart Center of Munich, State of Bavaria

### SUMMARY

In 1977, a report appeared from a conference of the Bavarian Academy of Occupational and National Medicine of Munich, with the title "Passive Smoking at the Work Place." In this report, a majority consensus was reached to the effect that passive smoking is indeed an annoyance, but not a health hazard for the non-smoker, and that legal regulation is not required. Since this report, various epidemiological-statistical studies have been published by means of which attempts have been made to demonstrate health injury to non-smokers by passive smoking. In the present work, the results of these works are critically reviewed. It has been established that these publications do not provide sufficient evidence to prove health injury from passive smoking. One must be cautious, therefore, about rushing into premature conclusions from these publications, or demanding legislative action because of them.

Key words: Smoking - Passive smoking - No health injuries

2023380404

Since the appearance of the report of the Bavarian Academy of Occupational and National Health of Munich in 1977 (1), passive smoking has been discussed in increasingly controversial terms. On the appearance of this report, the idea was accepted that passive smoking was indeed an annoyance, but injury to health could be ruled out. In the meantime, a series of publications have appeared in which an association of passive smoking with health injury is asserted. These investigations have led to a flood of further publications, in which the results of the originally reported work have been interpreted quite differently. Currently there is a reiterated impression that the health-injurious effects of passive smoking are already proved. Many of these publications have nothing to do with scientific argumentation. A hodge-podge of half and total falsehoods, polemics against those with other opinions, and unsubstantiated demands on legislators cannot, of course, help in answering the open question.

What is really the situation as to the new findings about passive smoking?

1) It is certain that cigarette smoke contains a large number of carcinogenic substances. Their distribution in main - and sidestream smoke is different from one substance to another. In the first place, the passive smoker breathes a highly diluted sidestream smoke, which corresponds, as a maximum, to the smoke from a fifth to a half cigarette per day (2). How much smoke the passive smoker actually takes into his lungs is unknown, since the passive smoker does not inhale more or less through

2023380405

the mouth, but rather inhales shallowly through the nose.

Besides other carcinogens in the sidestream smoke, nitrosamines, to which a few authors ascribe special significance, are also present (Schmidt (3), Remmer (4)). Schmidt asserts that the nitrosamine content reaches values in smoky indoor areas which correspond to as much as 30 cigarettes per hour. This is true, and is prone to provoke emotions; from the scientific point of view, however, this is only of secondary value:

a) The dimethylnitrosamine content of the mainstream smoke of a filter cigarette is between <0.1 and 6 ng, and is on the average 3 ng (5). Under unfavorable circumstances, as much as 90 ng dimethylnitrosamine per hour may be picked up. Despite this, the daily average dimethylnitrosamine quantity taken in by passive smoking would be far less than the average nutritional limit for dimethylnitrosamine of 1100 ng (6). Furthermore, there are additional sources of dimethylnitrosamine in our environment, which can be seen from the compilation of Lehnert (7).

b) The quantity of nitrosamines picked up as a result of passive smoking and nutritional intake are to be compared with the endogenous nitrosamine formation. The most recent investigations have shown that one should consider values of 16,000 to 30,000 ng (8). This would be many times greater than the burden from passive smoking.

c) There are probably additional non-volatile nitrosamines

2023380406

which are present in food. Little is known about the magnitude of the amount and its carcinogenic potential, since the necessary methods of determination have not yet been developed (9). What may come out of this area can only be surmised.

2) For the reevaluation of these data, it has been asserted for years that the carcinogenic effects of passive smoking should be unambiguously confirmed by animal experiments (Portheine (10), Schmidt (3)). Actually, animal experiments on passive smoking have not yet been carried out. As Lehnert (7) has pointed out, a contrary assertion - if an intentional misrepresentation is not assumed - can be ascribed only to a faulty evaluation of animal experiments made heretofore. The experimental animals in these experiments were exclusively exposed to passive smoking of mainstream smoke at a high concentration. On the other hand, passive smokers are exposed to sidestream smoke at a highly diluted concentration. It is disturbing that the cited authors, to whom these associations have often been referred, apparently cannot learn more from them. They probably will stay with their false concepts in the future.

3) Recently, epidemiological studies have been carried out which purport to show the injuriousness to health of passive smoking. These appear to me to be much overvalued as to the force of their evidence. Evidently the desire to impress the non-smoker, to whom analytical data mean little or nothing, comes to the fore.

2023380402

a) Schmidt (3) cites 27 works, from which it would clearly appear that the children of smoking parents suffer from inflammation of the respiratory passages more frequently than the children of non-smokers. He limited himself to fewer than half of the cited studies, while in the rest of the works the theme was either not investigated at all and only other studies were cited, or the association reported by Schmidt was not found. Investigations which arrive at a "clear" conclusion are not easily executed. Indeed, allowance for differences in social status of the parents, the living conditions, the genetic predisposition, etc., make the assessment of the isolated factor "passive smoking" almost impossible. Since the necessary standardization of the lung function of passive-smoking children is not checked, doubt as to the asserted association is present in addition (11).

b) That passive smoking causes deterioration in lung function, as maintained by White and Froeb (12), must now be regarded as completely unproved, since the communication by Gostomzyk (13) on the planning and organization of the study involved; the work of White and Froeb really can make no claim to be taken seriously, in the scientific sense.

c) That non-smoking wives of smokers, according to Miller (14), die on average four years earlier than non-smoking women married to non-smokers, is supported by agreement in Germany only by the "Medical Working Circle on Smoking and Health." Scientists were aware from the start that the calculation by Miller was

2023380408

contrary to the fundamental rules of statistics (15).

d) Whoever wishes to provide himself an impression of the validity of the work of Hirayama (16) and Trichopoulos et al. (17) should read the interview with Hirayama and the commentary by Lehnert in the MMW (18). It is shown that the two studies both show decidedly weak positions. Their results, therefore, can neither affirm nor deny an association of lung cancer with passive smoking. Further, it is noteworthy that Schmidt (3) cites only Hirayama and Trichopoulos, but does not mention the work of Garfinkel (19) and Chan (20), whose results do not speak for a connection between passive smoking and lung cancer. The New York epidemiologist Wynder is of the same opinion (21):

"While sidestream smoke does contain the same types of tumorigenic agents contained in the mainstream smoke, quantitative studies on the smoke components in a room suggest that the quantity, especially of particulate compounds, that could be inhaled by an individual would not suffice to provide a carcinogenic burden."

In summary, I come to the following judgement: The injuriousness to health of smoke is beyond doubt. Considering the dose-effect relationships, I do not consider it possible to proceed beyond this to a conclusion about the injurious health effects of passive smoking. This appears to be the case because of scientific studies proving the association between health injuries

2023380409

and passive smoking are not available; contrary to the assertion of Schmidt, animal experiments on the subject of passive smoking have not yet been published anywhere in the world. The studies of White and Froeb (12), Miller (14), Hirayama (16) and Trichopoulos et al. (17) do not withstand scientific criticism.

Despite this, the necessity of strengthening protection of the non-smokers appears quite understandable to me. Finally, there are enough people who feel annoyed by passive smoking, and who sometimes wait in vain for consideration from the smoker. There is, furthermore, the problem of the passive smoker's exposure to carcinogenic substances, for which threshold values cannot be stated. For me, the question is whether all of this suffices to have recourse to stern regulatory measures, with all their consequences for our society. Finally, passive smoking is only one of many stresses, and finally, there are in our total environment, carcinogenic substances in minimal concentrations with which mankind has apparently learned to live in the course of his ontogeny.

In my view, society must determine whether smoking in public should be limited. This determination, however, presupposes information which is not manipulated in a one-sided way, but is directed toward reality. The question as to the consequences of federal intervention must also be set forth. What other human modes of behavior should be regulated in the same way as smoking? To what extent should such measures be demanded of the citizen of a free society? When a few authors like Schmidt (3)

2023380410

and Remmer (4) have already decided, I hesitate.

Remarks at Revision

In a recent work by Feyerabend and coworkers (Feyerabend, C.T. Higenbottam, M.A.H. Russel: Nicotine concentrations in urine and saliva of smokers and non-smokers. Brit. med. J. 284 (1982) 1002-1004), the impression is created that non-smokers who are exposed to tobacco take in about as much nicotine, and thus also other tobacco smoke components, as smokers who smoke up to 3 cigarettes. These conclusions are based on measurements of the nicotine concentration in urine and saliva in a total of 138 subjects (non-exposed non-smokers, exposed non-smokers, smokers). These statements appear misleading to me. An analysis of the work brings out the following questions:

1. Why do the non-exposed non-smokers attain the nicotine values of smokers of up to 2 cigarettes? Is it conceivable that non-smokers, without seeing or smelling it, could inhale such a considerable amount of cigarette smoke? Are methodological errors not present?
2. How can it be that there are smokers with small nicotine excretion? Should they not rather be considered as passive smokers, since they surely do not inhale, but instead puff?
3. For a dose - and therefore a risk assessment, should

2023380411

not better average values be used? Considering all the subjects, one would then arrive at a dose relation: Passive smoker/Smoker of 1:50, which is also considered possible by other authors.

Prof. Dr. Helmut Schievelbein, Institute of Clinical Chemistry,  
the German Heart Center of Munich, State of Bavaria, Lothstr.  
II, D-8000 Munich 2.

References.

- 1 Bayerische Akademie für Arbeits- und Sozialmedizin München: Passrauchen am Arbeitsplatz. A.W. Gentner Verlag, Stuttgart 1977
- 2 Hugod, C., L.H. Hartenius und P. Aszrap: Int. Arch. Occup. Environ. Health 42 (1978) 21-29
- 3 Schmidt, F.: Off. Gewordk.-Wes. 44 (1982) 130-132
- 4 Kremser, G.: MMW 124, Nr. 4 (1982) 20-21
- 5 Brunnenmann, A.D., W. Finel und F. Aloser: Oncology 37 (1980) 217-223
- 6 Spiegelhelder, R., G. Eisenbrand und R. Preußmann: Oncology 37 (1980) 211-216
- 7 Lechner, G.: MMW 124, Nr. 4 (1982) 19-20
- 8 Ohshima, H. und H. Borisch: Cancer Res. 41 (1981) 3658-3662
- 9 Preußmann, R.: Special Lecture Meeting on Nitrene Compounds, Tokyo 2.10.1981
- 10 Porthmeier, F.: MMW 124, Nr. 8 (1982) 12-13
- 11 Lebowitz, M.D., D.B. Arnett und R. Knudson: Int. Symp. on Indoor Air Pollution Health and Energy Conservation Amherst/Mass 13.-16.10.1981
- 12 White, J.R. und M.F. Probst: New Engl. J. Med. 302 (1980) 720-723
- 13 Cossemryk, J.G.: MMW 124, Nr. 4 (1982) 11
- 14 Miller, G.M.: J. Breathing 41 (1978) 5-9
- 15 Low, E.A.: World Smoking and Health 3 (1978) G2
- 16 Hiroshima, T.: Brit. Med. J. 282 (1981) 183-185
- 17 Trichopoulos, D., A. Kalofotis, L. Sparros und Z. Mochalova: Int. J. Cancer 27 (1981) 1-4
- 18 Hiroshima, T., L. Corfinkel und G. Lechner: MMW 123, Nr. 40 (1981) 1480-1488
- 19 Corfinkel, L.: J. Natl. Cancer Inst. 66 (1981) 2061-2066
- 20 Chen, W.C.: MMW 124, Nr. 8 (1982) 16
- 21 Wynder, E.L.: Seminar in Resp. Med. 3 (3) (1982) 135-139

2023380412

## Gibt es wirklich wichtige neuere Befunde zum Passivrauchen?

H. Schivelbein

Institut für Klinische Chemie, Deutsches Herzzentrum München des Freistaates Bayern

### Zusammenfassung:

1977 erschien der Bericht über eine Tagung der „Bayerischen Akademie für Arbeits- und Sozialmedizin, München“ mit dem Titel „Passivrauchen am Arbeitsplatz“. In diesem Bericht wurde ein mehrheitlicher KonSENSUS dahingehend erzielt, daß das Passivrauchen zwar eine Belästigung, aber keine Gesundheitsgefährdung für den Nichtraucher darstellt und eine gesetzliche Regelung nicht erforderlich sei. Seit diesem Bericht wurden verschiedene epidemiologisch-statistische Untersuchungen publiziert, mit deren Hilfe gezeigt wird, eine gesundheitliche Schädigung des Nichtrauchers durch Passivrauchen nachzuweisen. In der vorliegenden Übersicht werden die Ergebnisse dieser Arbeiten kritisch referiert. Es wird festgestellt, daß diese Publikationen nicht ausreichend belegt sind, um den Nachweis der Gesundheitsschädlichkeit des Passivrauchens zu führen. Es wird davor gewarnt, vereilige Schlüsse aus diesen Publikationen zu ziehen oder gar aufgrund dieser Verhältnisse gesetzgeberische Maßnahmen zu verlangen.

**Key-Words:** Smoking – Passive smoking – No health injuries

Passivrauchen ist seit dem Erscheinen des Berichtes der „Bayerischen Akademie für Arbeits- und Sozialmedizin, München“ im Jahre 1977 (1) zunehmend kontrovers diskutiert worden. Beim Erscheinen dieses Berichtes wurde die Vorstellung akzeptiert, daß Passivrauchen zwar eine Belästigung darstellen kann, daß aber eine Gesundheitsschädlichkeit ausschließen ist. In der Zwischenzeit ist eine Reihe von Publikationen erschienen, in denen ein Zusammenhang zwischen Passivrauchen und einer Gesundheitsschädigung behauptet wird. Diese Untersuchungen haben zu einer Flut von weiteren Publikationen geführt, in denen die in den Originalarbeiten mitgeteilten Ergebnisse äußerst unterschiedlich interpretiert werden. Heute besteht vielfach der Eindruck, daß die gesundheitsschädigende Wirkung des Passivrauchens bereits nachgewiesen ist. Mit wissenschaftlicher Argumentation haben viele dieser Publikationen nichts mehr zu tun. Ein Sammelsurium von Halbwahrheiten, Polemiken gegen Andersdenkende und unsubstantiierte Forderungen an den Gesetzgeber kann nämlich die offenen Fragen nicht beantworten helfen.

Wie verhält es sich nun mit den neuen Befunden zum Passivrauchen wirklich?

- 1) Es ist gesichert, daß Zigarettenrauch eine große Anzahl von karzinogenen Substanzen enthält. Ihre Verteilung

auf Haupt- und Nebenstromrauch ist von Substanz zu Substanz verschieden. Der Passivraucher atmet in erster Linie stark verdünnten Nebenstromrauch ein, der pro Tag mengenmäßig maximal dem Rauch einer Fünfzigzigarette bis zu einer halben Zigarette entspricht (2). Wieviel Rauch beim Passivrauchen wirklich in die Lungen aufgenommen wird, ist unbekannt, weil der Passivraucher den Rauch nicht über den Mund mehr oder weniger tief inhaliert, sondern flach über die Nase einatmet.

Neben anderen Karzinogenen kommen im Nebenstromrauch auch Nitrosamine vor, denen einzelne Autoren eine besondere Bedeutung beimessen (Schmidt [3], Remmer [4]). Schmidt behauptet, daß die Nitrosaminaufnahme in vergualmten Innenräumen Werte erreicht, die dem Gehalt im Hauptstromrauch von bis zu 30 Zigaretten pro Stunde entsprechen. Dies ist zutreffend, eignet sich auch, Emotionen zu erzeugen, hat aber aus wissenschaftlicher Sicht nur einen untergeordneten Stellenwert:

- a) Der Dimethylnitrosaminengehalt liegt im Hauptstromrauch einer Filterzigarette zwischen < 0,1 und 6 ng und beträgt durchschnittlich 3 ng (5). Unter ungünstigen Bedingungen können pro Stunde durch Passivrauchen wohl bis zu 90 ng Dimethylnitrosamin aufgenommen werden. Trotzdem dürfte die durchschnittliche tägliche Dimethylnitrosaminaufnahme durch Passivrauchen weit unter der durchschnittlichen ernährungsbedingten Dimethylnitrosaminaufnahme von 1100 ng (6) liegen. Im übrigen gibt es in unserer Umwelt weitere Dimethylnitrosaminquellen, wie aus der Zusammenstellung von Lehnert (7) ersichtlich ist.
- b) Der vom Passivrauchen und der Nahrungsaufnahme abhängige Nitrosaminaufnahme steht die endogene Nitrosaminbildung gegenüber. Neueste Untersuchungen ergeben, daß mit einer täglichen In-vivo-Entstehung von 16000 bis 30000 ng zu rechnen ist (8). Dies würde um ein Vielfaches über der Belastung durch Passivrauchen liegen.
- c) Es gibt vermutlich weitere nichtflüchtige Nitrosamine, die in der Nahrung vorkommen. Über die Höhe der Belastung und ihre kanzerogene Potenz ist wenig bekannt, da die erforderlichen Nachweismethoden noch nicht entwickelt sind (9). Was auf uns auf diesem Gebiet zukommt, ist vorerst nur zu erahnen.

2023380413

- a) Um diese analytischen Daten aufzuweisen, wird seit Jahren behauptet, die krebsverzeugende Wirkung des Passivrauchens sei im Tierexperiment einwandfrei gesichert (Kurtheine [10], Schmidt [3]). In Wirklichkeit sind Tierexperimente zum Passivrauchen bis heute nicht durchgeführt worden. Wie Lehmers (7) ausführt, kann eine gegenteilige Behauptung – wenn man eine bewußte Falschdarstellung nicht unterstellt – nur auf eine falsche Einschätzung der Methodik bei den bisher durchgeführten Tierexperimenten hinweisen. Die Versuchstiere wurden bei diesen Experimenten ausschließlich mit Hauptstromrauch in hoher Konzentration passiv berauscht. Dagegen wird Passivraucher dem Nebenstromrauch in stark verdünnter Konzentration ausgesetzt. Störend ist nur, daß die zitierten Autoren, denen diese Zusammenhänge des öfteren mitgeteilt worden waren, offensichtlich nicht mehr dazulernen können. Sie werden wohl auch in Zukunft bei ihren falschen Behauptungen bleiben.
- b) Seit neuestem werden auch epidemiologische Untersuchungen angeführt, die die Gesundheitsschädlichkeit des Passivrauchens aufzeigen sollen. Diese scheinen mir in ihrer Aussagekraft stark überbewertet zu sein. Offensichtlich kommt darin der Wunsch zum Ausdruck, den Nichtraucher zu beeindrucken, dem analytische Daten wenig oder nichts bedeuten.
- c) Schmidt (3) zitiert 27 Arbeiten, aus denen klar hervorgehen soll, daß die Kinder rauchender Eltern häufiger an Entzündungen der Atemwege erkranken als Kinder von Nichtrauchern. Er hätte sich auf weniger als die Hälfte der Zitate beschränken sollen, weil in den restlichen Arbeiten das Thema entweder gar nicht untersucht wurde und nur andere Studien zitiert wurden oder der von Schmidt angegebene Zusammenhang nicht gefunden wurde. Untersuchungen, die zu einer „klaren“ Aussage kommen, sind nicht leicht durchführbar. Schon die Berücksichtigung der unterschiedlichen sozialen Stellung der Eltern, der Wohnverhältnisse, der genetischen Prädisposition usw. machen die Beurteilung des isolierten Faktors „Passivrauchen“ außerhalb unmöglich. Daß nach der sotwendigen Standardisierung die Lungenfunktion passivrauchender Kinder nicht eingeschränkt ist, muß ebenfalls Zweifel an dem behaupteten Zusammenhang aufkommen (11).
- d) Daß Passivrauchen zu einer Beeinträchtigung der Lungenfunktion führt, wie White und Froeb (12) behaupten, muß spätestens seit der Mitteilung von Gostomzyk (13) über Planung und Organisation der angeführten Studie als völlig unbewiesen angesehen werden; die Arbeit von White und Froeb hat nämlich keinen Anspruch, wissenschaftlich ernstgenommen zu werden.
- e) Daß nach Miller (14) nichtrauchende Ehefrauen von Rauchern im Durchschnitt vier Jahre früher sterben als Nichtraucherinnen, die mit Nichtrauchern verheiratet sind, ist in Deutschland nur bei dem „Ärztlichen Arbeitskreis Rauchen und Gesundheit“ auf Zustimmung geslopen. Wissenschaftlern ist von Anfang an bekannt gewesen, daß die Berechnung von Miller gegen die fundamentalen Regeln der Statistik verstößt (15).

d) Wer sich einen Eindruck von der Wertigkeit der Arbeiten von Hirayama (16) und Trichopoulos et al. (17) verschaffen will, lese das Interview mit Hirayama und den Kommentar von Lehners in der MMW (18). Es wird feststellen müssen, daß die beiden Untersuchungen entscheidende Schwachstellen aufweisen. Ihre Ergebnisse können deshalb einen Zusammenhang zwischen Lungenkrebs und Passivrauchen weder bestätigen noch verneinen. Im übrigen fällt auf, daß Schmidt (3) nur Hirayama und Trichopoulos zitiert, nicht aber auf die Arbeiten von Gorfinkel (19) und Chen (20) eingeht, deren Ergebnisse nicht für einen Zusammenhang zwischen Passivrauchen und Lungenkrebs sprechen. Der gleichen Meinung ist auch der New Yorker Epidemiologe Hynder (21):

„Nebenstromrauch enthält zwar die gleichen tumorigen Substanzen wie der Hauptstromrauch. Messungen in Räumen zeigen jedoch, daß die inhalierbaren Mengen, insbesondere der Substanzen der Partikelphase, nicht ausreichen würden, um eine kanzerogene Belastung darzustellen.“

Zusammenfassend komme ich zu folgender Beurteilung: Die Gesundheitsschädlichkeit des Rauchens steht außer Zweifel. Davon ausgehend auf die Gesundheitsschädlichkeit des Passivrauchens schließen zu wollen, halte ich unter Berücksichtigung der Dosiswirkungsverhältnisse für nicht möglich. Genauso scheint aber zu geschehen, da wissenschaftliche Arbeiten, die einen Zusammenhang zwischen Gesundheitsschäden und Passivrauchen beweisen, nicht vorliegen; denn entgegen der Behauptung von Schmidt sind Tierexperimentelle Untersuchungen zum Thema Passivrauchen bis heute nirgendwo auf der Welt publiziert worden. Die Arbeiten von White und Froeb (12), Miller (14), Hirayama (16) und Trichopoulos et al. (17) halten einer wissenschaftlichen Kritik nicht stand.

Trotzdem erscheint mir die Forderung, den Nichtraucherschutz zu verstärken, durchaus verständlich. Schließlich gibt es genug Menschen, die sich durch das Passivrauchen belästigt fühlen und die auf die Rücksichtnahme der Raucher manchmal umsonst warten. Es gibt darüber hinaus das Problem, daß Passivraucher kanzerogenen Substanzen ausgesetzt sind und daß für diese Schwellenwerte nicht angegeben werden können. Mir stellt sich nur die Frage, ob all dies ausreichen kann, um regulative Maßnahmen mit all ihren Folgen für unsere Gesellschaft zu ergreifen. Schließlich ist Passivrauchen nur eine von vielen Belastungen, und schließlich kommen in unserer Umwelt überall kanzerogene Substanzen in Minimalkonzentrationen vor, mit denen der Mensch im Lauf seiner Ontogenese offensichtlich zu leben gelernt hat.

Meines Erachtens muß unsere Gesellschaft entscheiden, ob sie das Rauchen in der Öffentlichkeit eingeschränkt haben will. Diese Entscheidung setzt aber eine Information voraus, die nicht einseitig manipuliert ist, sondern der Realität gerecht wird. Auch die Frage nach den Folgen eines staatlichen Eingriffs muß gestellt werden. Welche weiteren menschlichen Verhaltensweisen müßten in gleicher Weise

2023380414

wie das Rauchen reguliert werden? Wie weit sind solche Maßnahmen dem Bürger einer freien Gesellschaft zuzumuten? Auch wenn einzelne Autoren wie Schmidt (3) und Remmer (4) sich bereits dafür entschieden haben, ich zögere.

#### Literatur:

- 1 Bayerische Akademie für Arbeits- und Sozialmedizin München: Untersuchungen am Arbeitsplatz. A.W. Gentner Verlag, Stuttgart 1977
- 2 Nagel, C., L.H. Howkins und P. Astorg: Int. Arch. Occup. Enviro. Health 42 (1978) 21-29
- 3 Schmidt, F.: Öffl. Gesundh.-Wes. 44 (1982) 130-132
- 4 Remmer, G.: MMW 124, Nr. 4 (1982) 20-21
- 5 Brunnenmann, K.D., W. Fink und F. Moser: Oncology 37 (1980) 217-222
- 6 Spiegelhalder, R., G. Eisenbrand und R. Prugmann: Oncology 37 (1980) 211-216
- 7 Lehavot, G.: MMW 124, Nr. 4 (1982) 19-20
- 8 Oshima, H. und H. Borisch: Cancer Res. 41 (1981) 3658-3662
- 9 Prugmann, R.: Special Lecture Meeting on Nitros Compounds. Tokyo 2.10.1981
- 10 Pischinek, F.: MMW 124, Nr. 8 (1982) 12-13
- 11 Lebowith, M.D., D.B. Arnett und R. Kadmon: Int. Symp. on Indoor Air Pollution Health and Energy Conservation Amherst/Mass. 13.-16.10.1981
- 12 White, J.R. und M.F. Frosch: New Engl. J. Med. 302 (1980) 720-723
- 13 Gorenstein, J.G.: MMW 124, Nr. 4 (1982) 11
- 14 Müller, G.M.: J. Breathing 41 (1978) 5-9
- 15 Low, Z.A.: World Smoking and Health 3 (1978) (2)
- 16 Hirose, T.: Brit. Med. J. 282 (1981) 183-185
- 17 Trichopoulos, D., A. Kalandidi, L. Sparos und E. Neokleous: Int. J. Cancer 27 (1981) 1-4
- 18 Hirose, T., L. Corfakel und G. Lehavot: MMW 123, Nr. 40 (1981) 1480-1488
- 19 Corfakel, L.: J. Natl. Cancer Inst. 66 (1981) 1061-1066
- 20 Chen, W.C.: MMW 124, Nr. 4 (1982) 16
- 21 Hynder, E.L.: Seminar in Resp. Med. 3 (3) (1982) 135-139

#### Anmerkung bei der Korrektur:

In einer kürzlich erschienenen Arbeit von Feyerabend u. Mitarb. (Feyerabend, C., T. Higenbottam, M.A.H. Russel: Nicotine concentrations in urine and saliva of smokers and non-smokers. Brit. med. J. 284 [1982] 1002-1004) wird der Eindruck erweckt, daß Nichtraucher, die dem Tabakrauch ausgesetzt sind, etwa soviel Nikotin und damit auch andere Tabakrauchbestandteile aufnehmen können wie Raucher, die bis zu 3 Zigaretten rauchen. Diesen Schlußfolgerungen liegen Messungen der Nikotinkonzentration im Harn und Speichel von insgesamt 138 Probanden (nicht exponierten Nichtrauchern, exponierten Nichtrauchern, Rauchern) zugrunde. Mir scheint diese Aussage irreführend zu sein. Eine Analyse der Arbeit wirft folgende Fragen auf:

1. Aus welchen Gründen erreichen nicht exponierte Nichtraucher die Nikotinwerte von Rauchern von bis zu zwei Zigaretten? Ist es denkbar, daß Nichtraucher, ohne es zu sehen oder zu riechen, eine so beachtliche Menge Zigarettenrauch inhalieren? Liegen nicht doch methodisch bedingte Fehler vor?
2. Wie verhalten sich Raucher mit einer geringen Nikotinszufuhr wirklich? Sind sie nicht eher Passivraucher zuzurechnen, da sie sicher nicht inhalieren, sondern nur passen?
3. Sollten für eine Dosis- und damit auch Risikobeschätzung nicht besser Mittelwerte verwendet werden? Unter Berücksichtigung aller Probanden könnte man dann auf eine Dosisrelation Passivraucher/Raucher von 1:50, wie sie auch von anderen Autoren für möglich gehalten wird.

Prof. Dr. Helmut Schievelbein, Institut für Klinische Chemie, Deutsches Herzzentrum München des Freistaates Bayern, Lochstr. 11, D-8000 München 2

2023380415

## Concentration of DimethylNitrosamine in the Air of Smoke-Filled Rooms

G. STEHLIK, O. RICHTER, AND H. ALTMANN

*Österreichisches Forschungszentrum Seibersdorf, Institut für Biologie, A-2444 Seibersdorf, Austria*

*Received February 3, 1982*

In order to evaluate the contribution of volatile nitrosamines from tobacco smoke to indoor air pollution, *N*-nitroso-dimethylamine (NDMA) and *N*-nitroso-diethylamine (NDEA) were measured in indoor air under artificial and natural conditions. In controlled experiments under extreme conditions, we found that tobacco smoke-related NDMA levels above 0.07 ng/liter were associated with a highly irritating atmosphere which was scarcely tolerable to those present. In smoke-filled rooms under natural conditions NDMA levels ranged from 0.02 to 0.05 ng/liter, except a minimum value of <0.01 ng/liter in a restaurant and a maximum of 0.07 ng/liter in a dancing bar. These NDMA levels are thus below comparable values reported by others. The NDMA/NDEA ratios found in air samples taken from some rooms under conditions of everyday life are quite different from those found in sidestream smoke of cigarettes. Irritation was not reported under natural conditions. From the results it is concluded that NDMA levels, measured under real life conditions, are usually not caused by tobacco smoke alone. Evidence for other sources of volatile nitrosamines is discussed.

### INTRODUCTION

In 1977 the occurrence of volatile nitrosamines in sidestream smoke of cigarettes was reported (Brunnemann *et al.*, 1977). In 1978 Brunnemann *et al.* and Brunnemann and Hoffmann published measurements of NDMA concentrations in samples of air taken from smoke-filled rooms under natural conditions. They reported NDMA levels ranging from <0.003 to 0.13 ng/liter and an extreme value of 0.24 ng/liter in air from a bar. The authors relate this NDMA concentration to the cigarette smoke burden of the room.

In order to relate the irritating effect of cigarette smoke with the NDMA indoor air level due to tobacco smoke, a number of experiments were performed under artificial conditions. The subjects were questioned about their physical state in this series of studies. For comparison, some measurements under natural conditions were also carried out.

### MATERIALS AND METHODS

The chemicals used were of analytical grade (Merck, Darmstadt). Methylene chloride was distilled in a glass column before use. NDMA and NDEA were obtained from Eastman Kodak. The sampling was done using Miner pumps (MSA, Monitaire Samples) and two liquid traps (Wertheim washing flasks) each filled with 75 ml of a solution of 1 M citrate phosphate (pH 4.5) buffer/ascorbic acid (1.79 g) (Brunnemann *et al.*, 1977). The cleanup of the nitrosamine/buffer solution (Brunnemann *et al.*, 1977) as well as the confirmation by uv (Krull *et al.*, 1979) of the nitrosamine values was performed according to the literature. The recovery rate was assumed

2023380416

to be 80% in all cases. The values determined were corrected appropriately (Siehlík *et al.*, 1982).

The nitrosamines were analyzed by GC and determined by thermal energy analyzer (TEA). The following instruments were used under the indicated conditions.

**Varian 3700/TEA-502 (Integrator Autolab II)**

Oven temperature program: 60°C—10°/min—160°C

Injector temperature: 250°C

Carrier gas: Helium, 25 cm<sup>3</sup>/min

Separation column: Glass, 1.5 m × 1.75 mm i.d., 5% Ucon 50-HB-660 in Chromosorb W 120/140, pretreated according to Daniewsky and Aue (1978)

**Hp 5710/TEA-502 (Integrator HP 3380A)**

Oven temperature: 170°C, isotherm

Injector temperature: 220°C

Carrier gas: Helium, 35 cm<sup>3</sup>/min

Separation column: Glass, 3.5 m × 2 mm i.d., 10% Carbowax 20 M-TPA on Supelcopor 100/120.

The CO concentration was determined with an ir analyzer Miran 80 (Wilks).

In the experiments performed under artificial conditions, with no persons present in the room, blank values for NDMA and NDEA in the indoor air were determined before starting the experiment. The concentrations for both nitrosamines were below the detection limit of 0.01 ng/liter.

## RESULTS

### *Artificial Conditions*

The smokers present in the room were asked to smoke as much as possible. Doors and windows were predominantly kept closed during the experiment. For experiments 2 to 6, sampling was carried out during the 2-hr smoking period. For experiment 1 sampling was continued for an additional hour after the smoking period. For experiment 7 sampling was carried out during the 1-hr smoking period and for 2 hr afterward. The results are summarized in Table 1.

### *Natural Conditions*

For these experiments, the smoking behavior of the persons present was not influenced in any way. Sampling was carried out without knowledge of those present. People could leave the room and open doors and windows as they wished. The results are summarized in Table 2.

## DISCUSSION

Although our results and those of earlier investigations (Brunnemann *et al.*, 1977; Brunnemann *et al.*, 1978; Brunnemann and Hoffmann, 1978) show that volatile nitrosamines are detectable in indoor air, an estimation of the contribution of sidestream smoke cannot be given with the data available. Brunnemann *et al.* (1975) and Brunnemann and Hoffmann (1978) reported an NDMA level of 0.24 ng/liter.

2023380417

TABLE I  
MEASUREMENTS UNDER ARTIFICIAL CONDITIONS

Expt	Room size (m <sup>2</sup> )	Type of room	Number of persons present	Tobacco products consumed	Air volume collected (l)	NDMA (ng/l)	NDEA (ng/l)	CO (ppm)	Remarks
1	43	Conference room	11	64 cig. in 2 hr	571.4	0.07	b.d. <sup>a</sup>	n.m. <sup>b</sup>	Windows and doors were kept closed during the time of sampling. <sup>c</sup>
2	22	Office	3	35 cig. in 2 hr	600	0.13	b.d.	15 <sup>d</sup>	Windows and doors were not opened during the experiment. <sup>e</sup>
3	22	Office	3	38 cig. in 2 hr	600	0.15	b.d.	16 <sup>d</sup>	
4	22	Office	3	28 cig. in 2 hr	600	0.08	b.d.	n.m.	Windows were kept closed, the door was shortly opened <sup>f</sup> to four times during the experiment <sup>g</sup>
5	22	Office	2	12 cig. in 2 hr	600	0.02	0.01	8 <sup>d</sup>	
6	46	Office	7	18 cig., 4 pipes in 2 hr	600	0.08	b.d.	8 <sup>d</sup>	Windows were kept closed, the door was opened once for a short time. <sup>h</sup>
7	43	Conference room	10	40 cig. in 1 hr	617.1	0.022	b.d.	n.m.	Windows were kept closed, the door was opened several times. <sup>i</sup>

<sup>a</sup> b.d. = below detection limit of 0.01 ng/liter.

<sup>b</sup> n.m. = not measured.

<sup>c</sup> Reported irritation: very strong beyond tolerance limit.

<sup>d</sup> Maximum value.

<sup>e</sup> Reported irritation: none.

<sup>f</sup> Indicated irritation: very strong after 1 hr beyond tolerance limit; at this time the door was opened for the first time.

2023380418

TABLE 2  
MEASUREMENT UNDER NATURAL CONDITIONS.

Expt	Room size (m <sup>2</sup> )	Type of room	Number of persons present	Tobacco products consumed	Air volume collected (l)	NDMA (ng/l)	NDEA (ng/l)	CO (ppm)	Remarks
8	207	Working room	7	Continuous smoking (2 hr)	411.4 <sup>a</sup> 367.4	0.023 0.024	b.d. <sup>b</sup> b.d.	n.d. <sup>c</sup> n.d.	Windows were kept closed, doors were opened several times
9	301	Conference room	15	26 cig., 1 pipe, 6 cigarillos (2 hr)	411.4 367.4 <sup>a</sup>	0.029 0.033	b.d. b.d.	n.d. n.d.	Door was opened three times for a short time.
10	70	Office	6	27 cig. in 2 hr	600	0.03	0.03	9 <sup>d</sup>	Windows were kept closed, door was opened once.
11	50	Small conference room	12	37 cig., 4 pipes, 3 cigars (2 hr)	600	0.02	0.02	n.d.	Doors and windows were kept closed
12	120	Suburban restaurant	20	20-30 cig., 2 pipes (2 hr)	200	b.d.	b.d.	n.d.	
13	160	Restaurant in Vienna	23	20 cig. (1 hr)	207	0.01	b.d.	n.d.	
14	180	Restaurant in Vienna (Heuriger)	25	25-30 cig. (1 hr)	207	0.04	b.d.	n.d.	There was a wood-burning grill in the room with little smoke formation.
15	160	Restaurant in Vienna	23	15-20 cig. (1 hr)	207	0.05 <sup>d</sup>	b.d.	n.d.	
16	320	Dancing bar	30-70	Not to be determined, 4 hr collected	1200	0.07	0.2	n.d.	Room very smoky. <sup>e</sup>

<sup>a</sup>These values refer to samples from two sampling units which were analyzed separately.

<sup>b</sup>b.d. = below detection limit of 0.01 ng/l.

<sup>c</sup>n.d. = not determined.

<sup>d</sup>Maximum value.

<sup>e</sup>No indication of irritation was reported.

2023380419

a bar, which was allegedly due to tobacco smoke. In our experiments under artificial conditions (experiments 1-7), in which the persons were asked to smoke the maximum amount of tobacco products possible, the maximum value found was 0.15  $\mu$ g; NDMA/liter (experiment 3). The exposure conditions in these experiments (experiments 1-4 and 6) could seldom be found under normal living and occupational conditions. All the subjects concurred that they normally would have left the room long before the end of the experiment or opened windows and doors. As shown by experiment 4 (versus experiments 2 and 3) a short opening of the door lead to a drastic reduction of the NDMA concentration, even after heavy smoking.

Our measurements in smoke-filled rooms under natural conditions revealed NDMA levels normally between 0.02 and 0.05 ng/liter except a minimum value of <0.01 ng/liter for a restaurant and a maximum value of 0.07 ng/liter for a dancing bar. These levels are clearly below comparable values of Brunnemann *et al.* (1978) and Brunnemann and Hoffmann (1978), who reported NDMA levels ranging from 0.01 to 0.13  $\mu$ g/m<sup>3</sup> and an extreme value of 0.24 ng/liter for a bar. The unlikeliness of an NDMA level of 0.24 ng/liter being caused by tobacco smoke alone is obvious because irritation would have become intolerable.

According to analytical results of Brunnemann *et al.* (1980) and our own (1982), as well as measurements of irritating effects done by Weber *et al.* (1976), the limit of tolerance would be indicated theoretically by an NDMA level of 0.03-0.07 ng/liter caused by smoking alone, a range found for 23 different types of cigarettes. Further evidence for NDMA sources other than tobacco smoke appears when taking into consideration the NDMA/NDEA ratios. Data from the literature (Brunnemann *et al.*, 1977; 1980) as well as from our own investigations (1982) show that NDMA is present in the sidestream smoke of cigarettes, whereas NDEA, if any, is only detectable in negligible amounts. The amounts of NDEA found in a dancing bar (0.2 ng/liter, experiment 16) and in an office (0.03 ng/liter, experiment 10) are therefore, in our opinion, strong evidence for other sources of volatile nitrosamines in indoor air pollution.

Possibly part of the nitrosamines in indoor air may come from the outdoor atmosphere. Diesel engines (Yanysheva *et al.*, 1981; Goff *et al.*, 1980) as well as certain industries (Fine *et al.*, 1976; Fajen *et al.*, 1981) have been identified as nitrosamine emitters. Investigations by Fine *et al.* (1979) also show that rubber and leather products may contaminate indoor air with nitrosamines. Finally the NO<sub>2</sub> and NO concentration in the air is of importance for the formation of nitrosamines in the gas phase. Possible sources of nitric oxides are gas burners (Puxbaum, 1978; Yamazaki *et al.*, 1979) and unvented heaters (Nitta *et al.*, 1980).

The discrepancies in the available data on the nitrosamine burden of indoor air make evident the need for further investigations on this object. To get a more complete view of nitrosamine contamination, sources other than tobacco smoke have to be considered as well. To achieve this, one should refer to specific markers of emittants to clearly identify the sources.

#### REFERENCES

- BRUNNEMANN, K. D., YU, L., AND HOFFMANN, D. (1977). Assessment of carcinogenic volatile N-nitrosamines in tobacco and in mainstream and sidestream smoke from cigarettes. *Cancer Res.* 37, 3218-3222.  
BRUNNEMANN, K. D., ADAMS, J. D., HO, D. P. S., AND HOFFMANN, D. (1978). The influence of tobacco

2023380420

- smoke on indoor atmospheres. II. Volatile and tobacco-specific nitrosamines in main- and sidestream smoke and their contribution to indoor pollution. In *Proceedings, 4th Joint Conf. Sensing of Environmental Pollutants*, pp. 876-880. Amer. Chem. Soc., Washington, D.C.
- BRUNNEMANN, K. D., AND HOFFMANN, D. (1978). Chemical studies on tobacco smoke LIX. Analysis of volatile nitrosamines in tobacco smoke and polluted indoor environments. In *Environmental aspects of N-nitroso compounds* (E. A. Walker and L. Griciute, eds.), IARC Sci. Publ. No. 19, pp. 343-356. IARC, Lyon.
- BRUNNEMANN, K. D., FINK, W., AND MOSER, F. (1980). Analysis of volatile N-nitrosamines in mainstream and sidestream smoke from cigarettes by GLC-TEA. *Oncology* 37, 217-222.
- DANIEWSKI, M. M., AND AUE, W. A. (1978). Alternative method for bonding polymer layers to chromatographic supports. *J. Chromatogr.* 147, 119-125.
- FAJEN, J. M., ROUNBEHLER, D. P., AND FINE, D. H. (1981). Summary report on N-nitrosamine in the factory environment. Vortrag beim 7. In *International Meeting on Occurrence and Biological Effects on N-Nitroso Compounds*. Tokyo, IARC, Lyon (in press).
- FINE, D. H., ROUNBEHLER, D. P., SAWICKI, E., KROST, K., AND DEMARRAIS, G. A. (1976). N-Nitroso-compounds in the ambient community of Baltimore, Maryland. *Anal. Lett.* 9, 595-604.
- FINE, D. H., REISCH, J., AND ROUNBEHLER, D. P. (1979). Nitrosamines in new automobiles. N-Nitroso-compounds: Analysis, formation and occurrence. In *Proceedings, 7th International Symposium on N-Nitroso Compounds*. Budapest, 1979 (E. A. Walker, L. Griciute, M. Caregnaro, and M. Börzödy, eds.), pp. 541-554.
- GOFF, E. K., COOMBS, J. R., AND FINE, D. H. (1980). Determination of N-nitrosamines from diesel engine crankcase emission. *Anal. Chem.* 52, 1833-1836.
- KRULL, I. S., GOFF, E. U., HOFFMANN, G. G., AND FINE, D. H. (1979). Confirmatory methods for the thermal energy determination of N-nitroso compounds at trace levels. *Anal. Chem.* 51, 1706-1709.
- NITTA, H., YOKO HAMA, Y., MIURA, K., AOKI, S., AND MAEDA, K. (1980). Studies on personal exposure level of nitrogen dioxide: In case of housewives living in urban area. *J. Japan Soc. Air Pollut.* 15 (10), 418-425.
- PUXBAUM, H. (1978). Indoor air pollution by combustion sources. influence of the gas type an NO<sub>x</sub>-emission. Poster presented at Euroanalysis III, Dublin.
- STEHLIK, G., RICHTER, O., AND ALTMANN, H. (1982). Flüchtige Nitrosoamine im Nebenstromrauch von Zigaretten. In preparation.
- YAMANAKA, S., HIROSE, H., AND TAKADA, S. (1979). Nitrogen oxides emission from domestic kerosine-fired and gas fired appliances. *Atmos. Environ.* 13, 407-412.
- YANYSHEVA, NI. Y., CHERNICHONKO, I. A., LITVICHENKO, O. N., AND SOVERTKOVA, L. S. (1981). On the content of N-nitrosodimethylamine and its precursors in city air atmosphere. In *Carcinogenic Compounds and Their Precursors. Formation and Analysis in the Environment* (S. Bogovski, G. Loogna, and A. Itra, eds.), Abstracts of the 4th All-Union Symposium. Tallin. Inst. for Exp. and Clin. Med. Tallin, U.S.S.R.
- WEBER, A., JERMINI, C., AND GRANDJEAN, E. (1976). Irritating effects on man of air pollution due to cigarette smoke. *Amer. J. Public Health* 66, 672-676.

2023380421

Indoor & Ambient Air Quality  
1988

## NITROGEN DIOXIDE CONCENTRATIONS IN RESIDENCES

K.Soga & M.Pugac

Institute for medical Research & Occupational Health, M.Pijade 158, P.O.B.291, YU-4000 Zagreb, Yugoslavia

sts.

TM

due

### ABSTRACT

$\text{NO}_2$  concentrations were measured in kitchens and living rooms in a representative number of Zagreb homes. Possible influence on concentration levels of gas usage, the number of household members, their smoking habits, kitchen and living room volume, as well as their possible seasonal dependence, were investigated.

### INTRODUCTION

Nitrogen dioxide ( $\text{NO}_2$ ) is a by-product of high temperature combustion, and its main sources in ambient air are power generation stations, automobiles and industrial processes. It is an integral part of urban smog and plays a critical role in the complex of photochemical oxidant reactions (1). Main indoor  $\text{NO}_2$  sources are gas appliances, especially gas-cooking stoves. Peak values of  $\text{NO}_2$  concentrations occur during, or just after the operation of a gas stove. A model developed by Sexton and co-workers (1) shows that penetration from outdoors accounts for about 60% of concentrations, and that the average contribution from indoor sources, if they exist, equals about  $45 \mu\text{gm}^{-3}$  in the homes they have investigated. Personal exposure to  $\text{NO}_2$  is highly correlated with indoor concentrations, which is attributable to the fact that people spend a high percentage of time indoors. Quackenboss and co-workers (2) showed a significant difference in indoor  $\text{NO}_2$  concentrations between houses with gas appliances and those without them, as well as seasonal dependence of concentrations. Colome and co-workers (3) found that indoor  $\text{NO}_2$  concentrations ranged from 45 to 100% of outdoor levels, and that outdoor concentrations explained 13-36% of the cross sectional variability in indoor concentrations. Again they showed that gas appliances contributed significantly to increased indoor concentrations.

A pilot study performed by Ryan and co-workers (4) in a Boston residential area showed higher  $\text{NO}_2$  concentrations in homes with gas appliances than outdoors or in homes using only electricity. Those latter homes had lower concentration levels compared to outdoor levels. Concentrations measured in kitchens were higher than in living rooms, for both groups. In gas using households the main reason for this was a gas stove in the kitchen and in other

2023380422

homes that could have been a consequence of higher kitchen ventilation rates bringing in polluted outdoor air. Baker and co-workers (5) measured NO<sub>2</sub> concentrations in kitchens, bedrooms and outside of homes. They showed the impact of indoor sources, as well as the importance of improper installation, misuse, or inappropriate operation of gas appliances. At this Institute Pauković (6) investigated concentration levels of nitrogen oxides in ambient air, and their daily and seasonal rhythms. She found no significant seasonal difference in NO<sub>2</sub> levels against NO levels which were considerably higher in winter.

This paper deals with the results of a survey conducted in a representative number of Zagreb households. The aim of the survey was to examine NO<sub>2</sub> levels in Zagreb homes, to establish their relationship to a gas source, to the number of household members and to their smoking habits, and to determine their seasonal dependence.

#### EXPERIMENTAL PROCEDURE

NO<sub>2</sub> concentrations were measured in kitchens and living rooms of 86 Zagreb homes in winter and in 80 of these homes in the summer season. Household locations were spread all over the town and chosen randomly. Data on household members, their smoking habits, type of energy used for space heating and cooking, volumes of kitchens and living rooms were collected by use of a questionnaire. Measurements were performed with modified NO<sub>2</sub> passive samplers developed and tested at this Institute (7). Samplers were exposed to indoor concentrations for one week in each season.

#### RESULTS AND DISCUSSION

Table 1 shows the arithmetic mean, median, minimum and maximum concentrations and the concentrations which are not exceeded by 95% of the concentrations measured in the kitchens and living rooms for both sampling periods. Results for households with gas appliances and without them are shown separately. The households with gas appliances are placed in two subgroups, the ones connected to a gas pipeline and the others using gas cylinders. As the concentration distribution is asymmetric, the median tends to be more representative than the arithmetic mean. Table 2 shows the results of testing the significance of difference in concentration levels between different household groups for the winter and summer periods. A significant increase in concentration levels is noticeable in gas using households. There was no difference in NO<sub>2</sub> concentration levels between households connected to a gas pipeline and those using gas cylinders, except in kitchens in winter, when concentrations were significantly higher in households connected to gas pipelines. Concentrations in living rooms were higher too, but the difference cannot be considered significant. Kim (8) found different NO<sub>2</sub> concentrations in households using liquid gas and those using natural gas, especially in kitchens, but the difference was in the opposite direction compared to our findings. Significant correlations for all types of households, for winter and summer were found between NO<sub>2</sub> concentrations in kitchens and living rooms. Concentrations in living rooms tended to be lower indicating that the main NO<sub>2</sub> source might be located in the

2023380423

Table 1. Arithmetic mean (C), minimum ( $C_m$ ), maximum ( $C_M$ ), median ( $C_{50}$ ) and ( $C_{95}$ )  $\text{NO}_2$  concentrations during the winter and summer sampling periods in Zagreb homes (K - kitchen, L - living room) / $\mu\text{g m}^{-3}$ /

	Household group	N	C	$C_m$	$C_M$	$C_{50}$	$C_{95}$
	All	K 86 L 86	82 36	7 6	353 204	49 27	318 91
	Without gas	K 18 L 18	26 22	14 8	35 33	28 25	35 33
WINTER	With gas	K 68 L 68	97 39	7 6	353 204	59 29	326 120
	Pipeline	K 39 L 39	124 45	7 6	353 204	96 38	342 174
	Gas cylinders	K 27 L 27	62 33	16 10	326 141	52 29	110 60
	All	K 80 L 80	57 33	6 0	264 83	45 27	130 69
	Without gas	K 18 L 18	28 24	9 8	65 51	25 24	60 51
SUMMER	With gas	K 61 L 61	65 35	6 0	264 83	42 32	110 71
	Pipeline	K 33 L 33	74 36	6 0	264 83	56 33	107 82
	Gas cylinder	K 26 L 26	56 34	18 7	130 71	54 32	68 69

Table 2. Significance of difference in  $\text{NO}_2$  concentration levels among different household groups for the winter and summer periods (K - kitchen, L - living room)

		With gas	Pipeline	Gas cylinders
Without gas	W K	$< 10^{-6}$	$< 10^{-5}$	$< 0,001$
	L	$< 0,02$	$< 0,01$	$< 0,05$
	S K	$< 0,05$	$< 10^{-5}$	$< 10^{-5}$
	L	$< 0,02$	$< 0,05$	$< 0,01$
With gas	W K	-	-	-
	L	-	-	-
	S K	-	-	-
	L	-	-	-
Pipeline	W K			$< 0,01$
	L			-
	S K			-
	L			-

2023380424

kitchen. In winter, in households connected to gas pipelines a correlation was established between NO<sub>2</sub> concentrations in the kitchen and the number of household members and in summer time such correlation was found both in kitchens and living rooms. In households which used gas cylinders correlation did not exist.

Correlations between NO<sub>2</sub> concentration on the one hand and the number of smokers, the number of cigarettes smoked at home and kitchen and living room volumes, on the other were not found. No significant seasonal difference in NO<sub>2</sub> concentration levels was found for any type of household.

#### CONCLUSIONS

- NO<sub>2</sub> levels in Zagreb homes agree with the concentrations found in literature.
- Concentrations in kitchens and living rooms are highly correlated.
- In households using gas, concentrations in kitchens are significantly higher than in living rooms; meal preparation is thus concluded to be the main source of NO<sub>2</sub> in those households.
- No significant difference in NO<sub>2</sub> concentration levels was found between households using natural gas and those using gas cylinders, except for the winter period when concentrations were higher in the kitchens where natural gas was used.
- No significant seasonal differences in indoor NO<sub>2</sub> concentration levels were found.
- There is a correlation between the number of household members and NO<sub>2</sub> concentration levels in households using natural gas.
- There is no correlation between the number of smokers or the number of cigarettes smoked and NO<sub>2</sub> concentrations.
- There is no correlation between the kitchen or living room volume and indoor NO<sub>2</sub> concentrations.

#### ACKNOWLEDGEMENT

The authors wish to thank Mrs. Katarina Podeljak for her technical assistance.

#### REFERENCES

1. Sexton, K., Letz, R. and Spengler, J.D. (1983) *Environ. Res.*, 32, 151-166.
2. Quakenboss, J.J., Spengler, J.D., Kanarek, M.S., Letz, R. and Duffy, C.P. (1986) *Environ. Sci. Technol.*, 20, 775-783.
3. Colome, S.D., Wilson, A.L., Becker, E.W., Cunningham, S.J. and Baker, P.E. (1987) Proceedings of the 4th International Conference on Indoor Air Quality and Climate, Vol. 1, Institute for Water, Soil and Air Hygiene, Berlin, 405-409.
4. Ryan, P.B., Soczek, M.L. and Spengler, J.D. (1988) *J. Air Pollut. Control Assoc.*, 38, 22-27.
5. Baker, P.E., Cunningham, S.J. and Becker, E.W. (1987) Proceedings of the 4th International Conference on Indoor Air Quality and Climate, Vol. 1, Institute for Water, Soil and Air Hygiene, Berlin, 390-394.
6. Pauković, R. (1984) *Zašt. atm.*, 12, 59-62 (in Croatian).
7. Pauković, R. (1987) *Arh. hig. rada toksikol.*, 38, 17-23 (in Croatian).
8. Kim, Y.S., Spengler, J.D. and Yanagisawa, Y. (1986) *Environ. Int.*, 12, 401-406.

2023380425

## EFFECT OF CIGARETTE SMOKING ON RESIDENTIAL NO<sub>2</sub> LEVELS

Bennie W. Good, G. Vilcins, W. R. Harvey, D. A. Clabo, Jr., and A. L. Lewis  
Philip Morris, Inc., Research and Development, P.O. Box 26583, Richmond, Virginia 23261, USA

Two studies evaluating the levels and sources of nitrogen dioxide in approximately 90 employee homes in the Richmond area with continuous sampling during the weeks of August 5, 1980, and February 9, 1981, were performed using samplers in the living room, bedroom, kitchen, and outdoors. Additional data were collected concerning appliance usage, heating/cooling plant, ventilation and cigarette smoking. Results were analyzed using BMDP routines. The largest contributor to NO<sub>2</sub> concentration was found to be gas-fired kitchen appliances. The mean kitchen level for homes with gas appliances during the winter study was ~188 µg/m<sup>3</sup>. Excluding participants with gas kitchens, incremental influence due to cigarette smoking was detected. The 7-day, 3-room average level of NO<sub>2</sub> in the homes of nonsmokers and smokers without gas-fired appliances was 12 and 15 µg/m<sup>3</sup>, respectively, in the summer. The corresponding winter values were 19 and 22 µg/m<sup>3</sup>. Furthermore, the individual levels of NO<sub>2</sub> in the homes of smokers were generally below both the adjacent outdoor level and the National Ambient Air Quality Standard limit for annual exposure.

### Introduction

With Americans spending more than 90% of their time indoors, and with the move toward greater energy savings and thereby tighter residences and workplaces, there has been an increased emphasis on monitoring the levels of indoor air contaminants and correlating the levels with a variety of sources (Budiansky, 1980).

Nitrogen dioxide is corrosive, reactive and highly oxidizing, and may be toxic at high concentrations (Hueter *et al.*, 1973). Its half-life indoors is less than ½ that of CO (Wade *et al.*, 1975). Experiments at Philip Morris and elsewhere show that NO<sub>2</sub> results from oxidation of nitric oxide in aging cigarette smoke (Norman and Keith, 1965; Barkemeyer and Seehofer, 1968; Sloane and Kiefer, 1969; Vilcins and Lephardt, 1965).

In almost all published studies, a strong correlation has been shown between NO<sub>2</sub> levels and the presence and operation of gas cooking appliances (Spengler *et al.*, 1979; Hollowell, Budnitz and Traynor, 1977; Hollowell *et al.*, 1979; Eaton *et al.*, 1973). Although it is thought that cigarette smoking contributes to the NO<sub>2</sub> level, few researchers have demonstrated that relationship (Kasuga *et al.*, 1979; Weber and Fischer, 1980; Meyer *et al.*, 1981; Goldstein *et al.*, 1979).

Weber and Fischer (1980) investigated air pollution

due to tobacco smoke in 44 workrooms. The contribution to the level of NO<sub>2</sub> from smoking was claimed to be 45 µg/m<sup>3</sup>; more precisely, the measurement was the difference in NO<sub>2</sub> level between the unoccupied rooms at 4 to 6 a.m. and the occupied rooms. Since no room was totally free of smoke, it is difficult to assess the smoking contribution to the NO<sub>2</sub> level. The difference in levels of nicotine between unoccupied and occupied rooms was also measured; the results suggest only a +0.36 correlation with the corresponding NO<sub>2</sub> value, which questions how much of the 45 µg/m<sup>3</sup> is actually due to tobacco smoking. Weber and Fischer did not take into account the diurnal patterns of NO<sub>2</sub> (Hueter *et al.*, 1973). NO<sub>2</sub> is at minimum concentrations during nondaylight hours. As human activity, especially automobile traffic, increases in the hours just after dawn, the concentration of the primary contaminant, NO, increases. Then, as the ultraviolet energy from the sun becomes available, the NO<sub>2</sub> concentration increases (Hueter *et al.*, 1973).

A study by Goldstein *et al.* (1979) claims that each cigarette smoker in the home adds 16 µg/m<sup>3</sup> of NO<sub>2</sub>. However, their additive model is not well described and not without its own inconsistencies. For example, the same study reports a 62 µg/m<sup>3</sup> reduction in NO<sub>2</sub> for each flueless gas fire and 22 µg/m<sup>3</sup> reduction for each kerosene heater.

2023380426



Fig. 1. Labels with interleaved two-of-five barcode format used for sample tube identification.

The purpose of the present study was to measure the contribution of cigarette smoking to the NO<sub>2</sub> level in homes. Unfortunately, a true paired comparison between homes with and without cigarette smoking was difficult. Rather, volunteers were solicited from among our employees for participation, giving almost equal numbers of smokers and nonsmokers.

Sampling, which was performed using the Palmes personal sampler (Palmes *et al.*, 1976; Porter, 1981) for a 7-day average, was simple and inexpensive to perform for a large study. Sample analysis was further automated by using an AutoAnalyzer for determining the NO<sub>2</sub> concentration and a MINC-11 microcomputer for

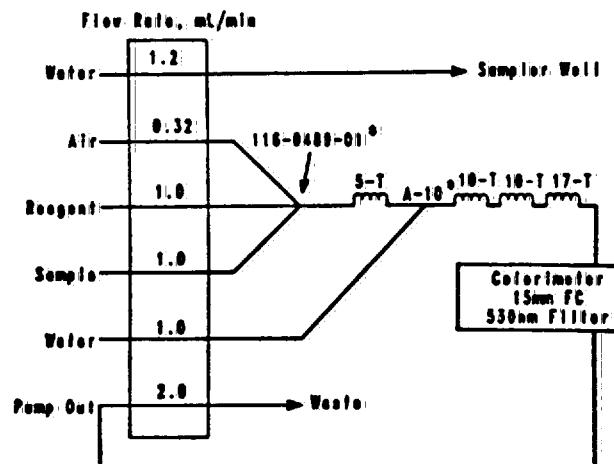


Fig. 2. AutoAnalyzer II configuration for the determination of NO<sub>2</sub>. Asterisk denotes Technicon part number.

Table 1. Details of BMDP file. (Units: a = number of days used; b = 1 if present, 0 if absent; c = absolute number of people; d = cigarettes smoked per week; e =  $\mu\text{g}/\text{m}^3$ ).

Variable	Description	Units
1-2	Name of participant	
3	Baseboard electric heat	a
4	Forced hot air-electric	a

Table 1. (Continued)

Variable	Description	Units
5	Electric heat pump	a
6	Kerosene space heater	a
7	Forced hot air-gas	a
8	Hot water radiator-gas	a
9	Forced hot air-oil	a
10	Hot water radiator-oil	a
11	Open fireplace	a
12	Fireplace with glass doors	a
13	Free-standing wood stove	a
14	Wood stove insert	a
15	Electric oven-summer	a
16	Gas oven-summer	a
17	Microwave oven-summer	a
18	Electric range-summer	a
19	Gas range-summer	a
20	Kitchen vent to outdoors-summer	a
21	Kitchen vent to indoors-summer	a
22	Electric water heater	b
23	Gas water heater	b
24	Oil water heater	b
25	Electric oven-winter	a
26	Gas oven-winter	a
27	Microwave oven-winter	a
28	Electric range-winter	a
29	Gas range-winter	a
30	Kitchen vent to outdoors-winter	a
31	Kitchen vent to indoors-winter	a
32	Central air conditioning	a
33	Window air conditioner	a
34	Attic fan	a
35	Ceiling or window fan	a
36	Auxiliary kitchen fan	a
37	Number of smokers in home-summer	c
38	Number of nonsmokers in home-summer	c
39	Number of cigarettes smoked-summer	d
40	Number of smokers in home-winter	c
41	Number of nonsmokers in home-winter	c
42	Number of cigarettes smoked-winter	d
43	NO <sub>2</sub> living room-summer	e
44	NO <sub>2</sub> bedroom-summer	e
45	NO <sub>2</sub> kitchen-summer	e
46	NO <sub>2</sub> outdoors-summer	e
47	NO <sub>2</sub> living room-winter	e
48	NO <sub>2</sub> bedroom-winter	e
49	NO <sub>2</sub> kitchen-winter	e
50	NO <sub>2</sub> outdoors-winter	e
51	Ratio NO <sub>2</sub> LR/outside-winter	
52	Ratio NO <sub>2</sub> BR/outside-winter	
53	Ratio NO <sub>2</sub> kitchen/outside-winter	
54	Difference NO <sub>2</sub> LR-outside-winter	
55	Difference NO <sub>2</sub> BR-outside-winter	
56	Difference NO <sub>2</sub> kitchen-outside-winter	
57	3-room ratio average-winter	
58	3-room difference average-winter	
59	Ratio NO <sub>2</sub> LR/outside-summer	
60	Ratio NO <sub>2</sub> BR/outside-summer	
61	Ratio NO <sub>2</sub> kitchen/outside-summer	
62	Difference NO <sub>2</sub> LR-outside-summer	
63	Difference NO <sub>2</sub> BR-outside-summer	
64	Difference NO <sub>2</sub> kitchen-outside-summer	
65	3-room ratio average-summer	
66	3-room difference average-summer	

2023380427

Table 2. Mean NO<sub>x</sub> levels in  $\mu\text{g}/\text{m}^3$  in homes with gas vs. electric kitchen appliances, comparing smokers vs. nonsmokers.

	Kitchen		Living Room		Bedroom	
	Summer	Winter	Summer	Winter	Summer	Winter
Electric (> 20 cigarettes)	15.6	21.3	16.5	23.5	14.0	21.3
Gas (> 20 cigarettes)	76.3	156.6	66.9	112.2	48.4	96.4
Electric (≤ 20 cigarettes)	11.8	20.3	12.4	19.6	10.7	17.5
Gas (≤ 20 cigarettes)	87.0	219.6	47.1	117.4	38.7	97.8

direct data collection and analysis. The majority of the data analysis, however, was performed on a host DEC-SYSTEM 20/60 using statistical routines from BMDP (Biomedical Computer Programs P-Series, Berkeley, CA).

### Experimental

A set of 12 sample tubes was prepared for each participant in the manner described in detail by Palmes

et al. (1981). The 12 tubes were tied together in groups of three for sampling principal areas in the home: living room, bedroom, kitchen, and outdoors. Each individual tube was labeled by sample number, location, and participant name. The sample number was also written as an Interleaved Two-of-Five bar code (Interface Mechanisms, Inc., Lynwood, WA) in order to facilitate acquiring and processing the data (Fig. 1). Additional tubes were prepared and labeled as blanks. These tubes remained closed in the lab during the sam-

Table 3. BMDP3D results comparing NO<sub>x</sub> levels ( $\mu\text{g}/\text{m}^3$ ) of smokers and nonsmokers without gas kitchen appliances. *P* is the probability of the observed difference in group means under the hypothesis that smoking has no effect on NO<sub>x</sub> levels.

	Summer		Winter	
	Nonsmoker	Smoker	Nonsmoker	Smoker
<b>Living room</b>				
Mean	12.4	16.5	17.5	21.3
Std. dev.	14.3	10.5	8.4	11.5
Sample size	54	38	49	38
Maximum	86.5	40.8	36.6	49.6
Minimum	-2.5	-0.4	5.7	-1.6
<i>P</i> = 0.13		<i>P</i> = 0.09		
<b>Bedroom</b>				
Mean	10.7	14.0	20.3	21.3
Std. dev.	11.7	9.4	9.5	11.1
Sample size	54	38	50	38
Maximum	66.9	42.7	57.1	54.3
Minimum	-2.7	-0.6	5.3	1.4
<i>P</i> = 0.16		<i>P</i> = 0.64		
<b>Kitchen</b>				
Mean	11.8	15.6	19.6	23.5
Std. dev.	12.5	10.0	8.1	13.2
Sample size	54	38	49	38
Maximum	72.0	44.7	44.5	65.6
Minimum	-3.4	0.5	5.7	1.4
<i>P</i> = 0.12		<i>P</i> = 0.12		
<b>Outside</b>				
Mean	21.3	22.6	52.3	50.0
Std. dev.	13.9	11.6	18.9	20.7
Sample size	54	38	48	35
Maximum	70.5	54.5	99.9	91.3
Minimum	1.1	6.3	18.7	8.9
<i>P</i> = 0.64		<i>P</i> = 0.62		

2023380428

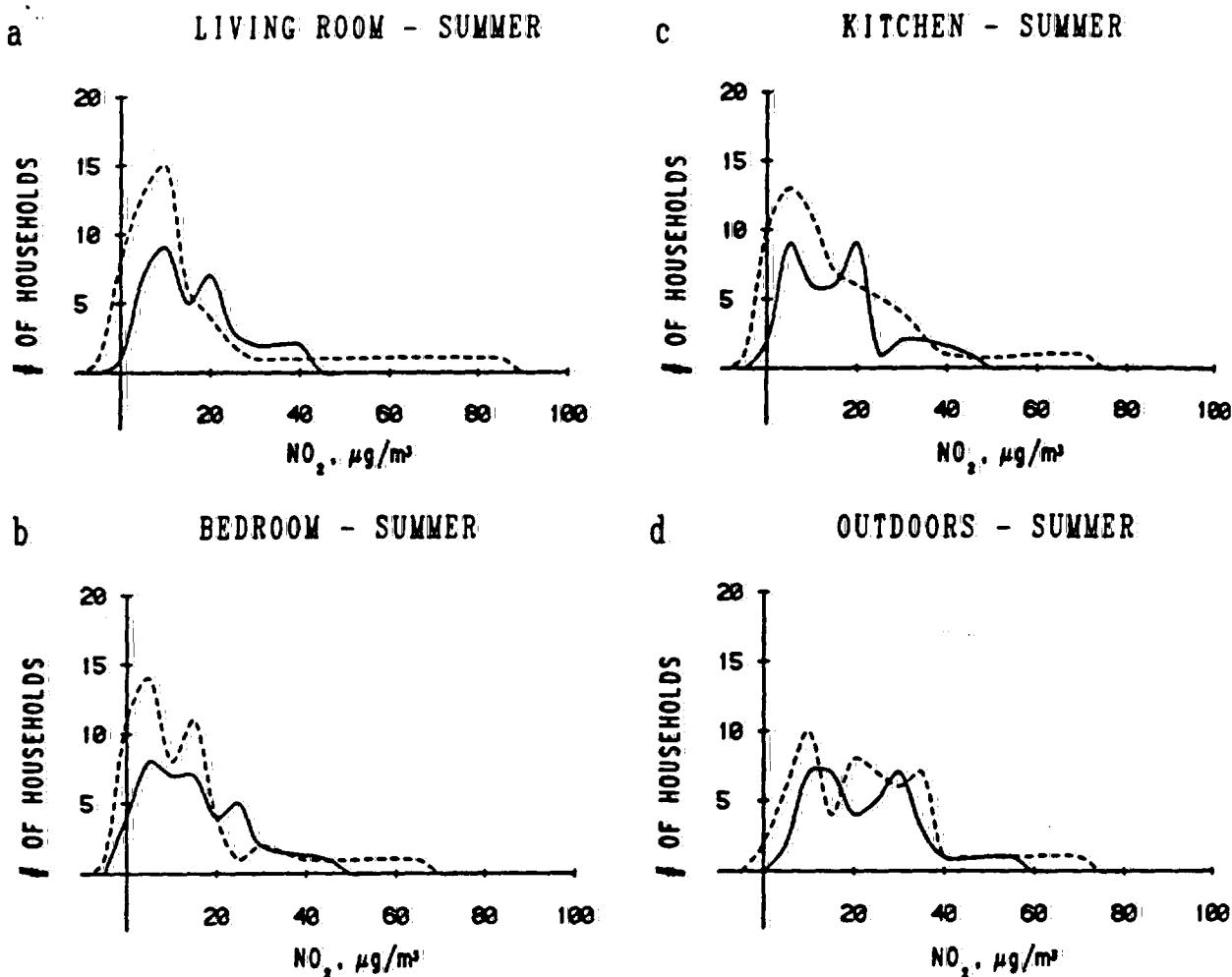


Fig. 3. Curves for the summer study showing the distribution of NO<sub>2</sub> levels ( $\mu\text{g}/\text{m}^3$ ) in the (a) living room, (b) bedroom, (c) kitchen, and (d) outdoors for smokers (—) and nonsmokers (----).

pling period. Participants were also given a log sheet to record the use of specific appliances, heating or cooling plant, and ventilation resources, as well as the number of cigarettes smoked in the home during the 7-day sampling period.

Following the 7 days, sample tubes were mixed with the blank tubes and analyzed in random order, to distribute possible systematic error in the analytical procedure among the samples. The bar codes eased data entry of the then random samples.

A Technicon AutoAnalyzer II system (Technicon Corp., Tarrytown, NY) was designed for the semiautomatic analysis of the NO<sub>2</sub> trapped on the triethanolamine coated grids. The sample tubes were extracted with deionized water and the extract submitted to the AutoAnalyzer. The analyzer was calibrated to measure NO<sub>2</sub> as nitrite colorimetrically via reaction with a sulfanilamide reagent (10 parts) and *N*-(1-naphthyl)-ethylenediamine dihydrochloride (1 part) (Fig. 2). The

reagent and five sodium nitrite standards were prepared daily. A DEC MINC-11/03 microcomputer (Digital Equipment Corp., Maynard, MA) was used to acquire and process the data. An Intermec Model 9300 bar code reader (Interface Mechanisms, Inc., Lynwood, WA) was connected to an ASCII port of the MINC to provide an alternative to typing the individual sample numbers. The sampling cam on the AutoAnalyzer, selected to give 1 sample/min, provided the time synchronization. The acquisition program, as well as a program that standardized peak heights and converted heights into ng of NO<sub>2</sub> per mL, were written in MINC BASIC. Following completion of the two programs, all sample identification numbers and their NO<sub>2</sub> values were transmitted to a DECSYSTEM 20/60 (Digital Equipment Corp., Maynard, MA) over an RS-232-C communication link.

Individual values were averaged for the same location. Final results were transcribed into a file compati-

2023380429

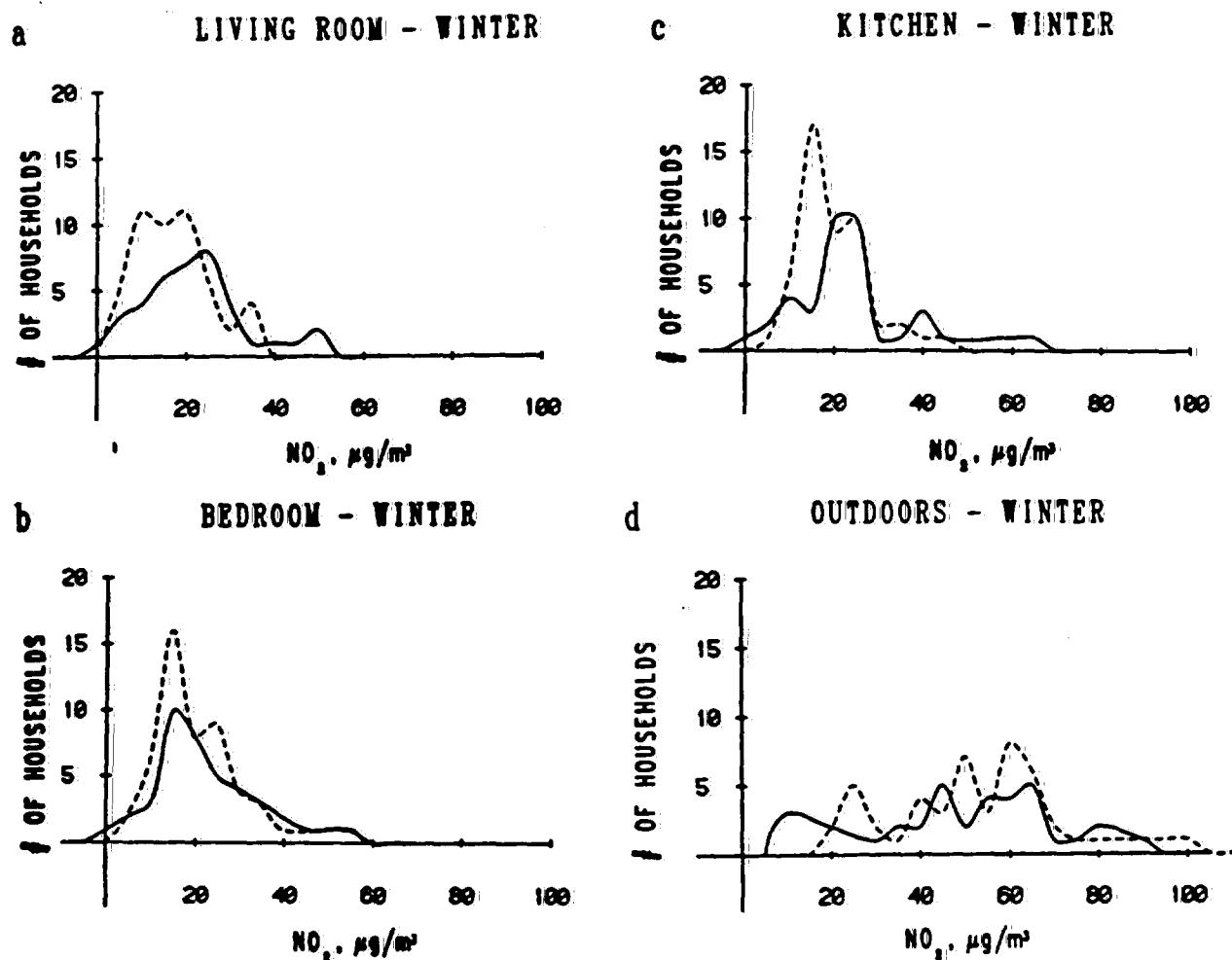


Fig. 4. Curves for the winter study showing the distribution of NO<sub>2</sub> levels ( $\mu\text{g}/\text{m}^3$ ) in the (a) living room, (b) bedroom, (c) kitchen, and (d) outdoors for smokers (—) and nonsmokers (---).

ble for BMDP input. Table 1 gives a detailed description of the data contained for each case, including the results of the logs provided by the participants and various mathematical transformations of the NO<sub>2</sub> results.

### Results and Discussion

There were three objectives in the analysis of the survey: (1) to identify the sources of nitrogen dioxide in the home, (2) to quantify these sources, and (3) to ex-

tract trends from the data which may result from cigarette smoking.

Table 5. Description of population for summer study.

Table 4. Geographical distribution of population.

	Smokers		Nonsmokers	
	Summer	Winter	Summer	Winter
Urban	8%	6%	7%	6%
Suburban	84%	80%	72%	77%
Rural	8%	15%	20%	17%

	Smokers	Nonsmokers
Number of participants	38	54
Mean no. of cigarettes smoked	115	2
Average no. nonsmokers in home	2.2	2.7
Average no. smokers in home	1.3	0.3
% use of window AC	29%	28%
% use of central air	58%	57%
% oil water heater	16%	6%
% gas water heater	16%	13%
% electric water heater	68%	81%
% kitchen vent-in	8%	15%
% kitchen vent-out	63%	57%
% electric range	100%	93%
% electric oven	97%	81%
% microwave oven	24%	11%

2023380430

*t*-tests

Using BMDP3D, which compares two groups by using *t*-tests, the means of NO<sub>2</sub> levels for smokers versus nonsmokers were obtained. This simple step proved the importance of attempting to make the two groups as equivalent as possible in terms of appliances, ventilation, and outside NO<sub>2</sub> level, leaving cigarettes as the only independent variable. Further comparisons between smokers and nonsmokers will classify a nonsmoker as one who smokes 20 cigarettes or less in the home during the 7-day period.

An illustration of the smoking and nonsmoking participants being unpaired involved the use of gas-fired kitchen appliances. It happened that 8 participants with gas kitchen appliances smoked more than 20 cigarettes, while only 3 smoked 20 or less. It is clear from Table 2, which contrasts smokers versus nonsmokers for cases with gas-fired kitchen appliances, that gas appliances would dominate our comparison between smokers and nonsmokers. Therefore, if these 11 cases are omitted, one can make a more valid measurement between smoking and nonsmoking participants. It should be noted that a probable cause for the high kitchen level in the winter for gas-appliance users was that several of them used range burners as auxiliary heat, resulting in an unvented gas space heater. In these cases, the National Ambient Air Quality Standard (NAAQS) for annual exposure (U.S. EPA, 1971) of 100 µg/m<sup>3</sup> of NO<sub>2</sub> was exceeded; although the level was substantially below the Threshold Limit Value (TLV) for an 8-h exposure of 10,000 µg/m<sup>3</sup> (American Conference of Governmental Industrial Hygienists, 1975).

Table 3 summarizes the results of the comparison of smokers versus nonsmokers without gas-fired kitchen appliances. The 3-room average of NO<sub>2</sub> for nonsmokers and smokers is 11.6 and 15.4 µg/m<sup>3</sup>, respectively, for the summer and 19.1 and 22.0 µg/m<sup>3</sup> for the winter. The outdoor difference between the groups (smokers minus nonsmokers) is 1.3 µg/m<sup>3</sup> in the summer and -2.3

Table 6. Description of population for winter study.

	Smokers	Nonsmokers
No. of participants	35	48
Mean no. of cigarettes smoked	102	2
Avg. no. of nonsmokers in home	2.0	2.7
Avg. no. of smokers in home	1.4	0.2
% electric heat	53%	40%
% oil heat	24%	50%
% gas heat	18%	17%
% wood heat	42%	33%
% oil hot water heater	11%	12%
% gas hot water heater	18%	17%
% electric hot water heater	71%	94%
% kitchen vent-in	5%	12%
% kitchen vent-out	61%	54%
% electric range	100%	98%
% electric oven	97%	100%
% microwave oven	24%	17%

µg/m<sup>3</sup> in the winter; these differences are smaller than would be expected by chance.

Figures 3 and 4 demonstrate the complexity of the comparison by showing the NO<sub>2</sub> distribution for the summer and winter, respectively. In all indoor cases, the smoking curve is shifted slightly to the right, while the mean outdoor level appears similar for the two groups. These figures illustrate that the comparison may not be as easy to interpret as Table 3 might suggest. A more complete description of the population involved is included in Tables 4-6.

*Multiple linear regression analysis*

BMDP1R estimates a least-squares regression equation between a dependent variable and one or more independent variables. The dependent variable we selected was variable 66 from Table 1 for the summer and 58 for

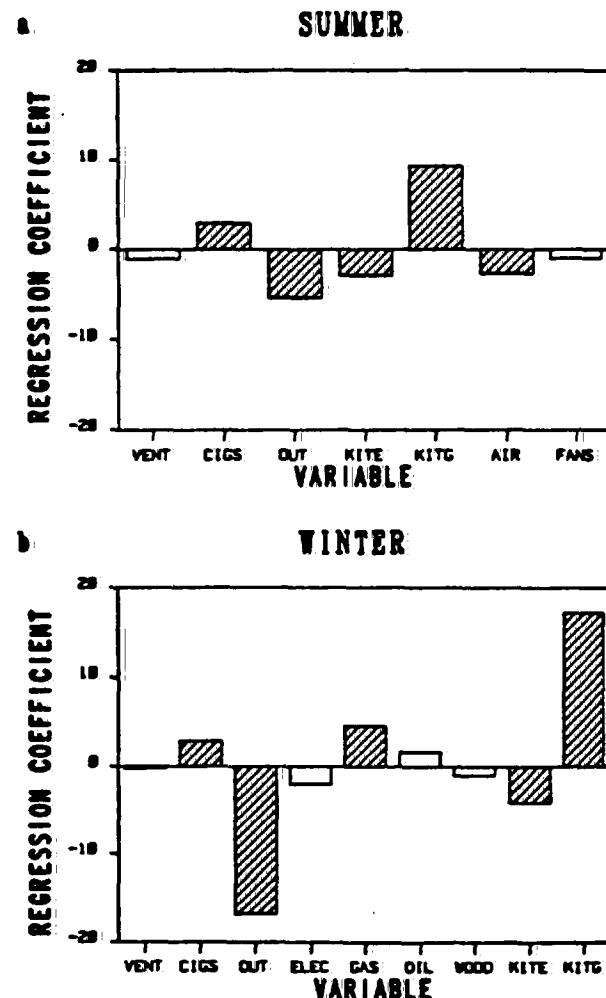


Fig. 5. Linear regression analysis coefficient results (a) for the summer and (b) for the winter. Dependent variables are: VENT, kitchen venting to outdoors; CIGS, number of cigarettes smoked in the house; OUT, the outdoor NO<sub>2</sub> level; KITE, electric kitchen appliances; KITG, gas-fired kitchen appliances; AIR, air conditioning; FANS, attic, ceiling, or window fans; ELEC, electric heat; GAS, gas heat; OIL, oil heat; and WOOD, wood heat. Shaded coefficients designate significance greater than 90%.

2023380431

the winter, the average of each of the indoor measurements minus the outdoor level. Independent variables for both summer and winter included all electric appliances, all gas-fired kitchen appliances, use of kitchen vent to the outside, outside NO<sub>2</sub> level, and the number of cigarettes smoked. Specific to the summer study, additional independent variables were air conditioning and ventilation. Winter specific independent variables were electric, gas, oil, and wood heat. All independent variables were standardized. The regression results are shown in Fig. 5 and detailed in Table 7. Coefficients with a level of significance less than 0.1 are shaded. The signs of the coefficients demonstrate either the contribution to or removal of NO<sub>2</sub>, or the correlation with other variables for the cases in which the variables are not truly independent. The use of a kitchen vent to the outside does seem to lower NO<sub>2</sub> levels, although the regression coefficient has a level of significance greater than 0.1. The larger vent coefficient in the summer versus the winter reflects the greater and, therefore, more effective use of vents in the summer. The outside air obviously provides a significant contribution to both studies; the outside air coefficient is negative because the dependent variable is the indoor NO<sub>2</sub> level minus the outdoor. Since the indoor level is generally lower than the outdoor, the dependent variables are generally negative; the outdoor regression coefficient seems to indicate protection from the outside air which the house offers.

The kitchen appliance regression coefficients illustrate that gas-fired appliances are the predominant term. The electric kitchen coefficient is negative, perhaps not due to its lowering of NO<sub>2</sub>, but because of its negative correlation with gas kitchens; i.e., a home would generally have either one or the other. Air conditioners significantly clean the inside air, while fans,

merely stirring the inside air or exchanging with the outside, do not have a significant effect on indoor levels. The cigarette coefficient is about  $\frac{1}{3}$  that of gas-fired kitchens in the summer study and  $\frac{1}{6}$  of that in the winter. Specific to the winter study, electric, oil, and wood heat did not make a significant contribution, while gas-fired furnaces did.

A correlation plot of predicted (or calculated) versus actual NO<sub>2</sub> levels is shown in Fig. 6, along with the theoretical line.

#### Factor analysis

BMDP4M is used to accept the entire data base (appliances used, heating/cooling, cigarettes smoked, NO<sub>2</sub> levels) of 28 summer and 35 winter variables and to extract from these, and rotate meaningfully, a lesser number of independent factors. A breakdown of the factors for both studies in terms of the amount of variance explained is shown in Fig. 7. In both cases the

Table 7. BMDP1R results (multiple linear regression) for the summer and winter. All independent variables are standardized. Coefficients are depicted in Fig. 5.

	Variables	Coefficients	t	p(2 tail)
Summer	Intercept	- 4.8		
R = 0.79	Kitchen vent	- 1.1	- 1.0	0.32
	Cigarettes	2.8	2.7	0.01
	Outside	- 5.5	- 4.8	0.00
	Electric kitchen	- 3.0	- 2.0	0.05
	Gas kitchen	9.3	6.4	0.00
	Air conditioning	- 2.7	- 2.4	0.02
	Fans	- 1.0	- 0.9	0.37
Winter	Intercept	- 24.9		
R = 0.87	Kitchen Vent	- 0.2	- 0.1	0.89
	Cigarettes	2.8	1.8	0.08
	Outside	- 16.9	- 9.5	0.00
	Electric Heat	- 1.9	- 0.8	0.46
	Gas Heat	4.5	1.7	0.09
	Oil Heat	1.6	0.6	0.54
	Wood Heat	- 1.0	- 0.6	0.53
	Electric Kitchen	- 4.1	- 1.9	0.06
	Gas Kitchen	17.2	8.1	0.00

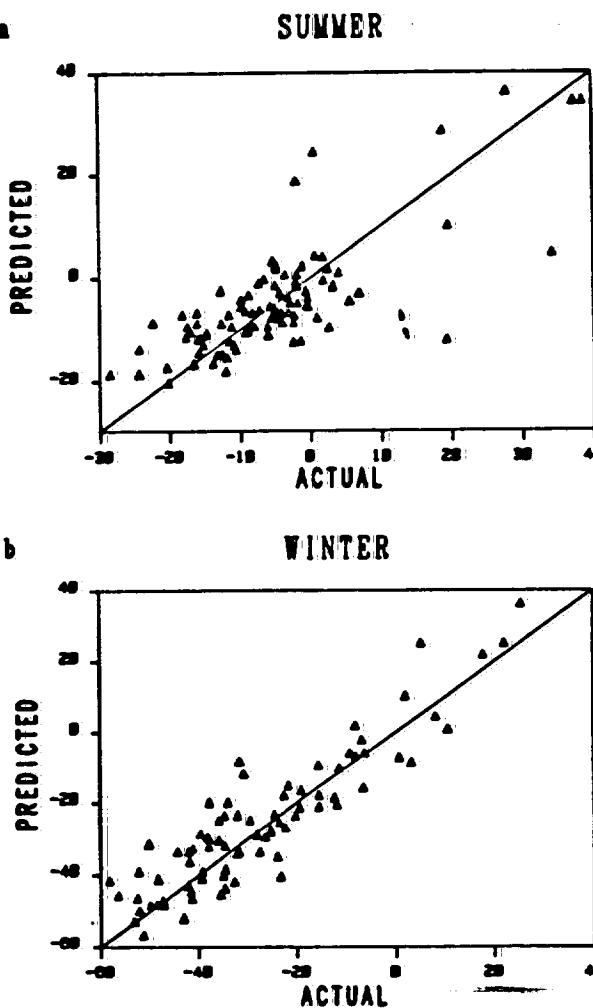
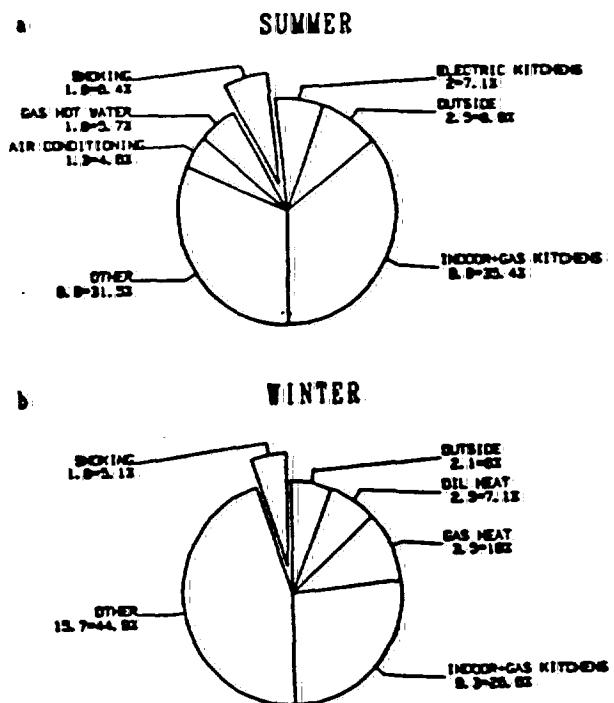


Fig. 6. Linear regression analysis plot of predicted (calculated) vs. actual NO<sub>2</sub> levels (a) for the summer and (b) for the winter. Triangles represent the individual cases. Correlation coefficient (R) equals 0.79 for the summer (a) and 0.87 for the winter (b).

2023380432

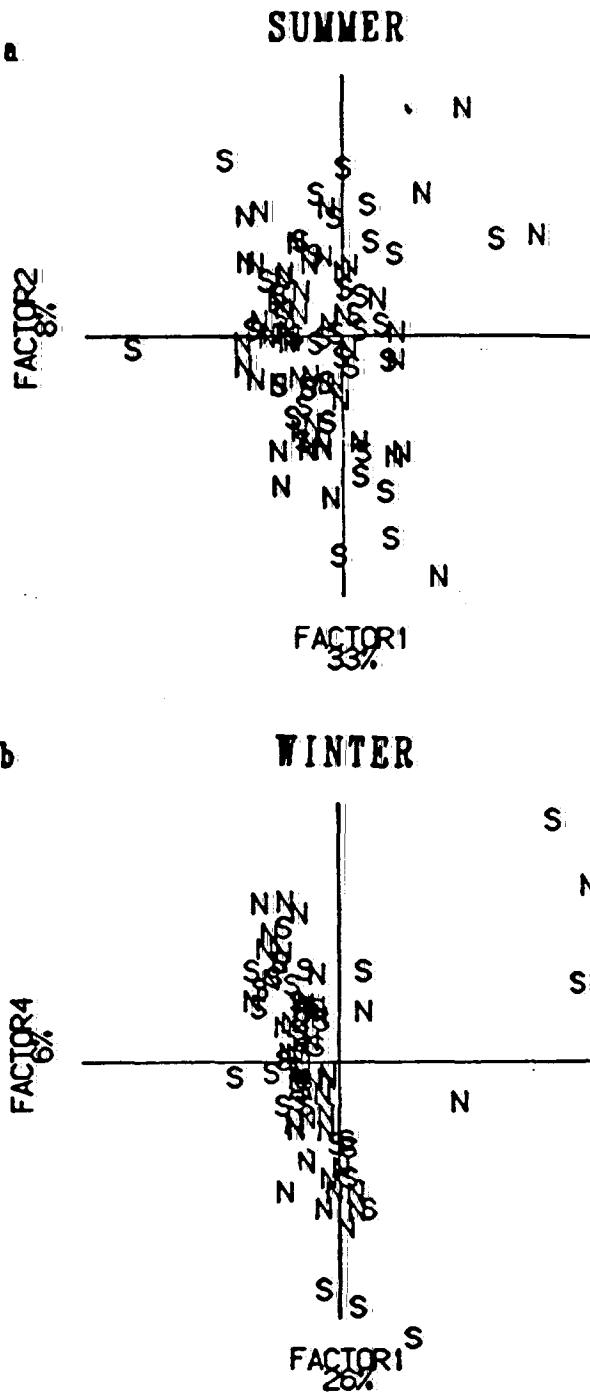


**Fig. 7.** Pie chart illustrating the percent variance of the entire data base explained by factors from BMDP4M for (a) summer and (b) winter. The smoking factor includes the number of cigarettes smoked and the number of smokers and nonsmokers in the home.

predominant factor was principally loaded, indicating coefficients of near 1.0, while coefficients of other variables are near 0, with all interior NO<sub>2</sub> levels plus gas kitchen appliances. When variables are together in a given factor, it indicates a high degree of interrelationship. The smoking factor, including the number of smokers and nonsmokers, accounts for 5%-6% of the variance of the entire data set (Table 1). This does not indicate a 5%-6% contribution to the indoor NO<sub>2</sub> by cigarettes, but rather, by its exclusion from factor 1, this demonstrates its failure to correlate with indoor levels at all. When we plot scores for factor 1 (inside and gas appliance kitchens) versus factor 2 (outside) for the summer study (Fig. 8a) and label smokers (> 20 cigarettes) and nonsmokers, it is difficult to differentiate the groups along the factor 1 axis, although the Ss appear slightly in the positive direction, which concurs with the *t*-tests results. A similar plot of the winter is shown in Fig. 8b, where differentiation of smokers and nonsmokers is less obvious. An example of the data extraction power of factor analysis is shown in Fig. 9, which plots factor 1 scores (indoor and gas appliance kitchens) against the smoking factor. The plot shows almost 100% differentiation.

## **Conclusions**

## A simple and inexpensive sampling procedure, semi-



**Fig. 8.** Factor scores for factors including indoor vs. outdoor NO<sub>x</sub> levels (a) for the summer and (b) for the winter. Cases that smoked 20 or more cigarettes are labeled S, and those that smoked less than 20 are labeled N.

automated data acquisition, and well-documented statistical methods were effectively combined to show that the increased level of  $\text{NO}_2$  due to cigarette smoking is barely measurable over a 7-day period. Furthermore, while the  $\text{NO}_2$  increment measured here for participants without gas-fired appliances is 4 and  $3 \mu\text{g}/\text{m}^3$  in the

2023380433

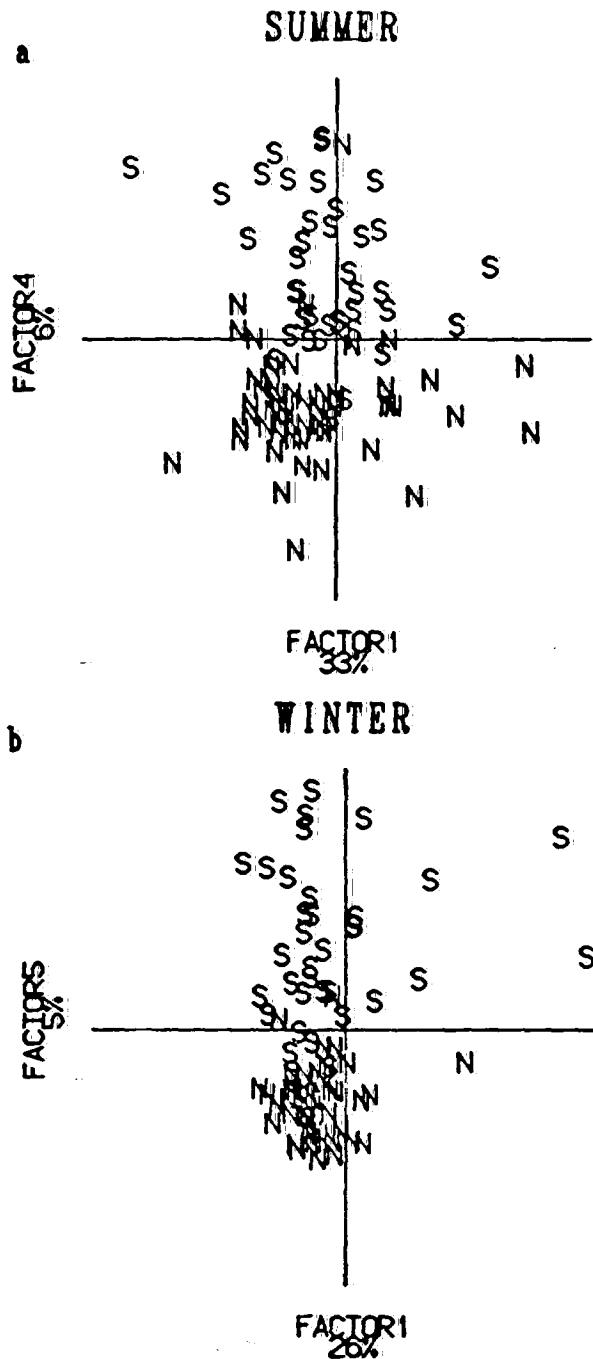


Fig. 9. Factor scores for factor including indoor NO<sub>x</sub> levels vs. the factor including the number of smokers, nonsmokers, and cigarettes smoked in the home during (a) the summer study and (b) the winter study. Cases are labeled as in Fig. 8.

summer and winter, respectively, the absolute levels are well below both the NAAQS of 100  $\mu\text{g}/\text{m}^3$  for annual exposure and the adjacent outdoor level. In addition, the contribution to the indoor NO<sub>x</sub> level due to gas-fired

kitchen appliances completely overpowers that due to the contribution from cigarette smoking.

**Acknowledgment**—The authors wish to thank Dorothy Clark and Wilton Suiter for their aid in sample tube preparation and analysis, and to Anne Donathan, Greg Kuhn, and Howard Clark for their help in the preparation of the manuscript.

#### References

- American Conference of Governmental Industrial Hygienists (1975) TLV's for chemical substances and physical agents in the workplace environment with intended changes for 1975. ACGIH, Cincinnati, OH.
- Barkemeyer, H. von and Seehofer, F. (1968) Zur untersuchung der Gas-Dampf-Phase des Cigarettenrauches, *Beitr. Tabakforsch.* 4, 278-282.
- Budiansky, S. (1980) Indoor air pollution, *Environ. Sci. Technol.* 14, 1023-1027.
- Eaton, W. C., Shy, C. M., Finkle, J. F., Howard, J. N., Burton, R. M., Ward, G. H., and Benson, F. B. (1973) Exposure to indoor nitrogen dioxide from gas stoves. U.S. Environmental Protection Agency, Research Triangle Park, NC.
- Goldstein, B. D., Melia, R. J. W., Chinn, S., Florey, C. duV., Clark, D., and John, H. H. (1979) The relation between respiratory illness in primary schoolchildren and the use of gas for cooking. II. Factors affecting nitrogen dioxide levels in the home, *Int. J. Epidem.* 8, 339-345.
- Hollowell, C. D., Berk, J. V., Lin, C., Nazaroff, W. W., and Traynor, G. W. (1979) Impact on energy conservation in buildings on health. LBL-9379, Lawrence Berkeley Laboratory, Berkeley, CA.
- Hollowell, C. D., Budnitz, R. J., and Traynor, G. W. (1977) Combustion-generated indoor air pollution. Presented at the Fourth International Clean Air Congress, Tokyo, Japan.
- Huetter, F. G., Goerke, H. W., Berksan, N. A., Liberti, A., and Ternisien, J. (1973) Air pollution: Air quality criteria for nitrogen oxides. EPA PB-240 575, U.S. Environmental Protection Agency, Washington, DC.
- Kasuga, H., Hasebe, A., Osaka, F., and Matsuki, H. (1979) Respiratory symptoms in school children and the role of passive smoking. *Tokai J. Exp. Clin. Med.* 4, 101-114.
- Meyer, M. B., Comstock, G. W., Tockman, M. S., Helsing, K. J., and Cohen, B. H. (1981) Respiratory effects of household exposure to tobacco smoke and gas cooking. Presented at the International Symposium on Indoor Air Pollution, Health and Energy Conservation, Amherst, MA.
- Norman, V. and Keith, C. H. (1965) Nitrogen oxides in tobacco smoke, *Nature* 205, 915-916.
- Palmer, E. D., Gunnison, A. F., DiMaggio, J., and Tomczyk, C. (1976) Personal sampler for nitrogen dioxide, *Am. Ind. Hyg. Assoc. J.* 37, 570-577.
- Porter, G. (1981) Personal monitors of pollutants. *Dimensions/NBS* 65, 9-12.
- Sloane, C. H. and Kiefer, J. E. (1969) Determination of NO and NO<sub>x</sub> in cigarette smoke from kinetic data, *Tob. Sci.* 13, 180-182.
- Spengler, J. D., Ferris, Jr., B. G., Dockery, D. W., and Spizer, F. E. (1979) Sulfur dioxide and nitrogen dioxide levels inside and outside homes and the implications on health effects research, *Environ. Sci. Technol.* 13, 1276-1280.
- U.S. Environmental Protection Agency (1971) National Ambient Air Quality Standards, *Fed. Reg.* 36, 8187.
- Vilcins, G. and Lephardt, J. O. (1975) Aging processes of cigarette smoke: Formation of methyl nitrite, *Chem. Ind.* 22, 974-975.
- Wade III, W. A., Cote, W. A., and Yocom, J. E. (1975) A study of indoor air quality, *J. Air Pollut. Control Assoc.* 25, 933-939.
- Weber, A. and Fischer, T. (1980) Passive smoking at work, *Int. Arch. Occup. Environ. Health* 47, 209-221.

2023380434

# **PRESENT AND FUTURE OF INDOOR AIR QUALITY**

**Proceedings of the Brussels Conference,  
14-16 February 1989**

*Editors:*

**C.J. Bieva, Y. Courtois and M. Govaerts**



**1989**

**EXCERPTA MEDICA, Amsterdam – New York – Oxford**

2023380435

© 1989 Elsevier Science Publishers B.V. (Biomedical Division)

All rights reserved. No part of this publication may be reproduced, stored in a retrieval system or transmitted in any form or by any means, electronic, mechanical, photocopying, recording or otherwise without the prior written permission of the publisher, Elsevier Science Publishers B.V., Biomedical Division, P.O. Box 1527, 1000 BM Amsterdam, The Netherlands.

No responsibility is assumed by the Publisher for any injury and/or damage to persons or property as a matter of products liability, negligence or otherwise, or from any use or operation of any methods, products, instructions or ideas contained in the material herein. Because of rapid advances in the medical sciences, the Publisher recommends that independent verification of diagnoses and drug dosages should be made.

Special regulations for readers in the USA - This publication has been registered with the Copyright Clearance Center Inc. (CCC), 27 Congress Street, Salem, MA 01970, USA. Information can be obtained from the CCC about conditions under which photocopies of parts of this publication may be made in the USA. All other copyright questions, including photocopying outside the USA, should be referred to the copyright owner, Elsevier Science Publishers B.V., unless otherwise specified.

International Congress Series No. 860  
ISBN 0 444 81134 6

*This book is printed on acid-free paper.*

*Published by:*

Elsevier Science Publishers B.V.  
(Biomedical Division)  
P.O. Box 211  
1000 AE Amsterdam  
The Netherlands

*Sole distributors for the USA and Canada:*

Elsevier Science Publishing Company Inc.  
655 Avenue of the Americas  
New York, NY 10010  
USA

Printed in The Netherlands.

2023380436

## FOREWORD

Indoor air quality conservation and procedures for the measurement of related potential pollutants, such as radon, asbestos, gases, pesticides, tobacco smoke and bacteria from air conditioning systems, have seen important changes in recent years, while the range and the scope of the studies have continued to expand.

In addition to helping preserve public health, the field of interest is now extending to include such areas as architectural design, ventilation engineering, sociology, psychology and legal aspects. Related analytical techniques like gas chromatography and mass spectroscopy have undergone parallel refinements and their range of application has broadened.

These advances were discussed at the Conference 'Present and Future of Indoor Air Quality', held in Brussels, February, 1989, following symposia on indoor air quality at Essen and Tokyo in 1987 and London in 1988. The sessions were attended by about 200 scientists representing 20 countries. A total of 92 papers and posters were presented covering such topics as pathogenesis and epidemiology, sources of indoor air contamination and risk assessment, chemistry of indoor air related to the outdoor air quality, social and psychological aspects of poor indoor air quality, motivation and attitudes, future guidelines for the improvement of indoor air quality through architectural and ventilation design, and air quality monitoring.

The proceedings include full texts and posters presented during the meeting. The organising committee hopes that they will constitute a useful guide for the improvement of our indoor air quality in the future.

2023380437

# CONTENTS

## OPENING SESSION: RISK ASSESSMENT AND HEALTH

Cancer control and lifestyle medicine <i>E.L. Wynder</i>	3
Sick building syndrome - Clinical aspects and prevention <i>C. Molina</i>	15
The ambient air, perceptions and tolerance thresholds: Changing social standards <i>N. Delruele</i>	23
Concerns and risks of home pollution assessed from the 329 cases reported to a Poison Control Centre during 1987 <i>G. Riboulet-Delmas, R. Garnier and M.-L. Efthymiou</i>	29
Carbon monoxide poisoning: Epidemiology and prevention <i>B. Tissot, M. Govaerts and N. Tinant</i>	37
Current state of research on air quality inside buildings <i>J.C. Loewenstein</i>	43
Radon and lung cancer: A case-control study in southern Belgium <i>A. Poffijn, R. Mak, P. Weynants, M. Vanhoorne and J. Prignot</i>	51
Indoor radon in Belgium <i>A. Poffijn and H. Vanmarcke</i>	55

## SECTION 1: ETS AND PASSIVE SMOKING

Environmental tobacco smoke and lung cancer: Is it the smoke or the diet?  
*L.C. Koo*

65

Dietary habits are of limited importance in influencing the lung cancer risk among Japanese females who never smoked  
*T. Hirayama*

77

An example of extra-poisson variation suggesting an under-specified model  
*S.J. Kilpatrick*

83

Lung cancer risk by oral exposure  
*R. Rylander, H.-J. Haussmann and F.J. Tewes*

91

Passive smoking: Acute effects on lung function of sensitive persons  
*B. Danuser, A. Weber and A.L. Hartmann*

101

Evaluation of exposure to ambient tobacco smoke: A few concrete examples concerning the value of biological and atmospheric indicators  
*A.M. Laurent, Y.A. Courtois, S. Pretet, J.R. Claude and B. Festy*

103

2023380438

**Exposure to environmental tobacco smoke in the non industrial workplace under different conditions of ventilation and smoking regulation**

<i>T. Sterling, C. Collett and J. Ross</i>	111
<b>Passive smoking and lung cancer; fact or fiction?</b>	
<i>P. Lee</i>	119
<b>Initiatives taken within parents in school to reduce passive smoking risks</b>	
<i>C. Arciti, B. Doglio and L. Santi</i>	129
<b>Health care and society: Environmental tobacco smoke and lung cancer</b>	
<i>E.L. Wynder and G.C. Kabat</i>	135
<b>Isoprene - A potential indoor indicator for environmental tobacco smoke.</b>	
<i>G. Löfroth</i>	147
<b>The passive smoking myth.</b>	
<i>N. Mantel</i>	155
<b>Relative lung cancer risk from exposure to mainstream and sidestream smoke particulates</b>	
<i>J.J. McAughay, J.N. Pritchard and A. Black</i>	161
<b>Measurement of environmental tobacco smoke in an air-conditioned office building</b>	
<i>C.J. Proctor, N.D. Warren and M.A.J. Bevan</i>	169
<b>Nicotine and indoor air pollution</b>	
<i>C. Arfi, J. Kaloustian, A.M. Pauli, J. Pastor, F. Grimaldi, F. Gouezo and A. Viala</i>	173
<b>Assessing the validity of a Japanese cohort study</b>	
<i>M.W. Layard and J.R. Viren</i>	177

**SECTION 2: INDOOR AIR QUALITY: CHEMISTRY AND BIOLOGICAL EFFECTS**

**Exposure to ETS and its biological effects: A review**

<i>F.X. Adlkofer, G. Scherer, L. Von Meyerinck, Ch. Von Maltzan and L. Jarczyk</i>	189
<b>Carbonyl compounds in mainstream and sidestream cigarette smoke</b>	
<i>H. Schlitt and H. Knöppel</i>	197
<b>Sensory responses to environmental tobacco smoke from cigarettes that heat but do not burn tobacco</b>	
<i>J.C. Walker, R.A. Jennings, W.T. Morgan, J.H. Robinson, D.W. Griffith and J.H. Reynolds IV</i>	207
<b>Role of combustion products in bronchial hyperreactivity - A case control study</b>	
<i>M. Bert, J. Kühr, A. Hendel-Kramer, T. Urbanek, E. Schultz, H. Steiger and W. Karmaus</i>	215
<b>The importance of endotoxin and glucan for symptoms in sick buildings</b>	
<i>R. Rylander, S. Sørensen, H. Goto, K. Yuasa and S. Tanaka</i>	219

2023380439

An epidemiological study on dose-response relationship between B(a)P concentrations in indoor air and lung cancer mortality in Xuan Wei, China <i>He Xingzhou, Chen Wei and Chen Hong</i>	227
Health consequences of atopy, food intolerance and indoor environment: A case study <i>E. Sammaljärvi</i>	235
Assessment of exposure to emissions from a liquefied petroleum gas stove used in urban India <i>V. Haraprasad and J.M. Dave</i>	239
Quality of indoor environment in dwellings according to performance of heating system <i>D. Petráš</i>	243
Oxides of nitrogen and respiratory illness. Is there a susceptible population? <i>J.M. Sneddon and T.M. Bearpark</i>	247
The effect of indoor air pollution on health from coal burning containing high fluorides <i>Ji Rongdi and Cao Shouren</i>	253
Effect of reservoir fuel levels on emissions from a kerosene cook stove <i>J.M. Dave and V. Haraprasad</i>	257
Study of the small-scale relations between indoor and outdoor pollution (SO <sub>2</sub> - NO <sub>x</sub> - particles) in a hospital in the Paris region <i>J.C. Loewenstein</i>	261
Nicotine absorption in humans following exposure to environmental tobacco smoke generated from different types of cigarettes <i>J.D. deBethizy, L.E. Bates, R.A. Davis, D.L. Heavner, P.R. Nelson, J.C. Walker and J.H. Robinson</i>	269
Characterization of the environmental tobacco smoke generated by different cigarettes <i>P.R. Nelson, D.L. Heavner and B.B. Collie</i>	277
Health and social problems of carbon monoxide poisoning from the usage of anthracite coal in Korea <i>Yong Chung, Dongchun Shin and Youngwook Lim</i>	283
On the use of environmental tobacco smoke component ratios <i>G.B. Oldaker III, W.E. Crouse and R.M. Depinto</i>	287

### **SECTION 3: INDOOR AIR QUALITY: VENTILATION, CLIMATISATION AND POLLUTANTS**

Ventilation, health and energy conservation – A workable compromise <i>G. Robertson</i>	293
Ventilation system retrofits as a method of solution for the 'sick building syndrome' <i>C. Collett and E. Sterling</i>	301

2023380440

A few studies of air-conditioned environments <i>A. Mouilleseaux, F. Squinazi and B. Festy</i>	309
Regression model for indoor concentrations of combustion-generated gases <i>A. Hendel-Kramer, J. Kühr, R. Urbanek, H. Staiger, E. Schultz and E. Karmaus</i>	315
A Czechoslovak survey about ventilation habits in dwellings <i>L. Piršel</i>	321
Emissions from materials; The role of additives in PVC <i>K. Saarela, K. Kaustia and A. Kiviranta</i>	329
Influence of indoor air pollution on NO <sub>2</sub> personal exposure levels of schoolchildren <i>Y. Shimizu, S. Imai and Y. Tsunetoshi</i>	337
Indoor pollution by mineral fibers <i>J. Bignon, G. Dufour, M.A. Billon-Galand, A. Gaudichet, H. de Cremoux and P. Brochard</i>	347
The Acarex/Acarosan concept, its possibilities in allergology and dermatology <i>J.E.M.H. Van Bronswijk, G. Schober and F.M. Kniest</i>	355
Elimination of house dust mite excreta, the carriers of allergens in the indoor air <i>E. Bischoff, A. Fischer and B. Liebenberg</i>	363
Portable air sampler for measurements in aircraft and public buildings <i>J.F. van der Wal</i>	371
Destruction of acarids and biodecontamination of domestic environment <i>M. Blanc and Ch. Boutin</i>	379
Dust in the work environment: Effect of pharmacological agents on the airway response to inhaled cotton dust <i>P.J. Nicholls, M. McDermott, M. Bevan and D. Griffiths-Johnson</i>	385
Moulds, mites and moisture: A preliminary report on six cases of fungal damage in dwellings <i>H.S.M. Kort, J.E.M.H. Van Bronswijk and G. Schober</i>	389
Absolute indoor air humidity and the abundance of allergen producing house dust mites and fungi in the Netherlands <i>G. Schober</i>	395
Air quality control of public car parks <i>F. Cupelin and J.Cl. Landry</i>	401
<b>SECTION 4: INDOOR AIR QUALITY: PREVENTION AND FUTURE GUIDELINES</b>	
Demand controlled ventilating systems: New concepts for indoor air quality and energy conservation <i>L. Trepte</i>	407
A database of problem buildings: Learning by past mistakes <i>C.W. Collett, E.M. Sterling, T.D. Sterling and J.J. Weinkam</i>	413

2023380441

Treatment programme for microbiologically contaminated airconditioning installations <i>E. Clement and R. De Koster</i>	421
Present and future exceedance of health related reference values for air pollutants in Dutch dwellings. <i>H.J. Van de Wiel and E. Lebret</i>	427
French incentive actions for research on indoor air quality in buildings <i>J.-P. Marie, J. Dubois and P.-C. Jacquignon</i>	435
A nitrogen oxide analyser designed for the study of indoor pollution: The APPI-AC35 <i>B. Millancourt</i>	443
Automatic analyser prototype for atmospheric isoprene and monoterpenes <i>M.L. Riba, B. Clement, M. Haziza and L. Torres</i>	449
Air tightness, permeability or transparency to air in buildings, different names for one same problem. <i>M. Kilberger</i>	453
<b>Index of authors</b>	461

2023380442

## INFLUENCE OF INDOOR AIR POLLUTION ON NO<sub>2</sub> PERSONAL EXPOSURE LEVELS OF SCHOOLCHILDREN

YOSHIHARU SHIMIZU

Department of Public Health, Saitama Medical College, Moroyama-cho, Irima-gun,  
Saitama Prefecture, Japan

SHUJI IMAI

Department of Public Health, Osaka Prefectural Government, Japan

YOSHIZO TSUNETOSHI

Department of Public Health, Miyazaki Medical College, Japan

NOTICE  
This material may be  
protected by copyright  
law (Title 17 U.S. Code)

### SUMMARY

The study was conducted to clarify the influence on NO<sub>2</sub> personal exposure levels of indoor air pollution caused by passive smoking and heating apparatus, and outdoor air pollution, and the following results were obtained.

- (1) NO<sub>2</sub> personal exposure levels in winter, when heating apparatus were used, were almost twice as high as in other seasons.
- (2) Average levels of personal exposure broken down by heating apparatus type revealed approximately twice as high values for heating apparatus without a ventiduct than for those with a ventiduct.
- (3) When heating apparatus were not used, NO<sub>2</sub> personal exposure levels for the group with family smokers registered slightly higher values than for those with no family smokers. When heating apparatus were used, these differences disappeared. Of course, no influence of family smokers on the NO<sub>2</sub> personal exposure level was observed both in heating and non-heating periods.
- (4) A predicted equation to estimate NO<sub>2</sub> personal exposure levels was arrived at through multiple regression analysis. NO<sub>2</sub> personal exposure levels were designated as the objective variable, while ambient air pollution levels, use of heating apparatus without a ventiduct, housing structure, passive smoking and climatic conditions (temperature) were designated as explanatory variables. Application of this predicted equation to other areas outside the scope of the original study resulted in a significant correlation between the predicted and the observed values.

### OBJECTIVE OF STUDY

The study was conducted to clarify the influence on NO<sub>2</sub> personal exposure levels of indoor air pollution caused by family smokers and heating apparatus used in the home, and outdoor air pollution. Personal NO<sub>2</sub> exposure levels of subjects chosen from

2023380443

elementary schools in five areas with differing atmospheric pollution levels and group NO<sub>2</sub> exposure levels were measured using an NO<sub>2</sub>-filter badge.

## SUBJECTS AND METHODS

The study was conducted over a two year period from September 1985 to March 1987. Subjects for the study were all members of the 5th and 6th grade classes of five elementary schools which had a monitoring station within the school area. Each subject was asked to pin the NO<sub>2</sub> filter badge to their clothing for a length of 24 hours on one weekday per month so that we could measure personal NO<sub>2</sub> exposure levels and outdoor NO<sub>2</sub> levels. Details were also recorded concerning the presence of any smokers in the home on the day of the survey, the type of heating apparatus used and the frequency of its usage and the housing structure.

The five areas covered by the study are described in Fig. 1 as areas A to E. The survey period for areas A and C were as follows:

- Area A surveyed on two occasions:  
October 1985 to February 1986 (5 months) and September 1986 to February 1987 (6 months)
- Area C surveyed on one occasion:  
September 1986 to July 1987 (11 months)

Table 1 shows the period of the study and the number of schoolchildren surveyed by school. Subjects pinned the NO<sub>2</sub> filter badges to their chest over a 24 hour period on one day per month. Using this method we measured the personal NO<sub>2</sub> exposure levels and also simultaneously took readings of NO<sub>2</sub> levels in various areas by placing a filter badge in the classrooms, corridors, schoolyards and monitoring stations of each area.

Based on the results of the simultaneous survey on presence of family smokers, type of heating apparatus used and the frequency of use, we designated the heating period as being between November and March, and the remainder of the year as the non-heating period.

As can be observed in Table 1, the survey was performed on a total of 39 occasions - 25 times in the heating period and 14 times in the non-heating period. The reason for conducting more surveys in the heating period was that personal NO<sub>2</sub> exposure levels were likely to be influenced more by indoor air pollution variables in the heating period than in the non-heating period. The total number of surveys made over the three year period was 5,385.

## RESULTS

Table 2 shows the correlation coefficients between NO<sub>2</sub> measurements which were obtained with the filter badges placed in the outdoor monitoring stations, classrooms, corridors and schoolyards.

2023380444

Table 2-(1) shows a high positive correlation between outdoor NO<sub>2</sub> levels and corridor and schoolyard NO<sub>2</sub> levels for the entire survey period including both the heating and non-heating periods. Table 2-(2), which refers to the non-heating period, shows a high positive correlation between outdoor NO<sub>2</sub> levels and NO<sub>2</sub> levels of classroom, corridor and schoolyard. For the heating period, shown in Table 2-(3), while a high positive correlation coefficient was observed between outdoor NO<sub>2</sub> values and the schoolyard NO<sub>2</sub> values, a low coefficient was observed between outdoor and the classroom NO<sub>2</sub> values. This is due to the influence of the indoor heating apparatus used during the heating period.

It is of interest to note that the correlation coefficient between the measured NO<sub>2</sub> value using the NO<sub>2</sub> filter badge in the monitoring station and the value measured by the Saltzman method in the same station was very high --namely 0.975 as shown in Fig. 2.

Table 3 displays the mean value of NO<sub>2</sub> personal exposure levels by area and month of survey. The NO<sub>2</sub> personal exposure levels in each area over the November to March heating period were high compared to those in the non-heating period; in fact the figure was almost double. In addition, the figures by area show that the mean value of NO<sub>2</sub> personal exposure levels tends to be higher for both heating and non-heating periods in the area where the higher NO<sub>2</sub> values are measured at the monitoring station.

Table 4 shows the arithmetic mean of NO<sub>2</sub> personal exposure levels by type of heating apparatus and presence of family smokers in each area over the September to February period. This indicates that in every area the NO<sub>2</sub> personal exposure level for those family groups using heating apparatus without a ventiduct was almost double that of those who used apparatus with a ventiduct. However, no clear difference was detected between the groups exposed to passive smoking and those not exposed.

Table 5 shows the arithmetic mean of NO<sub>2</sub> personal exposure levels this time using the housing structure as the explanatory variable. The results show that there was no significant difference in NO<sub>2</sub> personal exposure levels according to the housing structure.

Multiple regression analysis including stratified factor was used to examine effects of various potential factors on NO<sub>2</sub> personal exposure levels.

NO<sub>2</sub> personal exposure levels was designated as the objective variable, while ambient NO<sub>2</sub> level, type of heating, housing structure, use of cookery gas apparatus in the kitchen and family smokers were used as explanatory factors.

For the ambient NO<sub>2</sub> level, the filter badge placed in the monitoring station was used. Each of the stratified factors were assigned a value of "1" (one) if they were thought to increase personal exposure levels and a value of "0" (zero) if this was not believed to be so, as shown in notes of Table 6.

Results of the analysis as shown in Table 6 suggest that for the non-heating period, the regression coefficients of outdoor NO<sub>2</sub> level and presence of family smokers were

2023380445

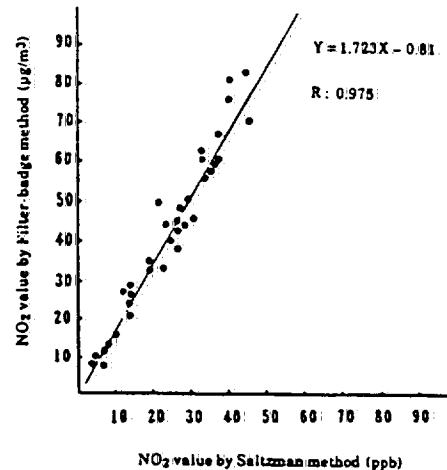
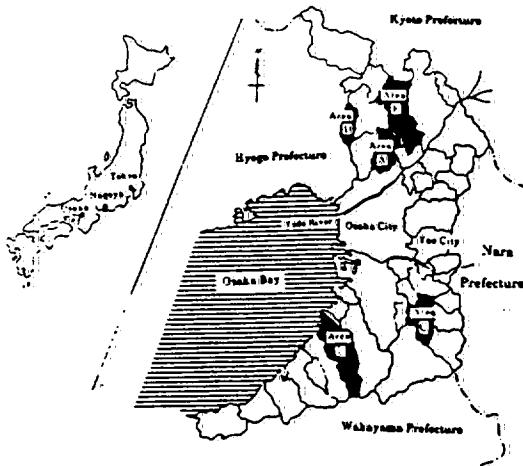


Fig. 2 Comparison of NO<sub>2</sub> values between Saltzman method and Filter-badge method

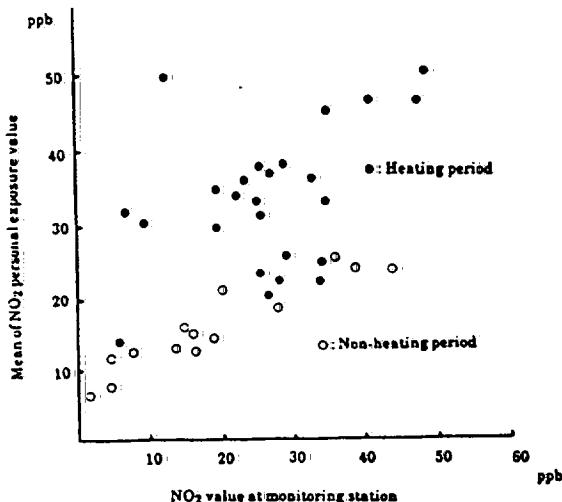


Fig. 3 Relations between mean of NO<sub>2</sub> personal exposure value and NO<sub>2</sub> values at monitoring station

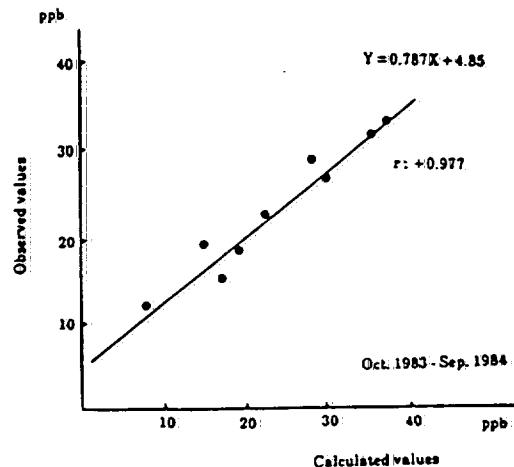


Fig. 4 Comparison of observed and calculated values of NO<sub>2</sub> personal exposure  
(Schoolchildren in Yao City)

Table 1 Area, number, period of survey and ambient NO<sub>2</sub> value at monitoring station

Year or survey	Survey area	Period of survey	Frequency of survey	Number of surveys	NO <sub>2</sub> value (at monitoring ST)
1985	A	Oct. 1985 - Feb. 1986	5(4)	195	0.029
	B	Oct. 1985 - Feb. 1986	5(4)	104	0.012
1986	A	Sep. 1986 - Feb. 1987	6(4)	188	0.029
	C	Sep. 1986 - July 1987	11(5)	117	0.014
1987	D	Sep. 1987 - Feb. 1988	6(4)	160	0.015
	E	Sep. 1987 - Feb. 1988	6(4)	173	0.026

Total man-days: 5,393

( ): Heating period when heating apparatus is used

2023380446

Table 2-(1) Correlation coefficient between NO<sub>2</sub> value for outdoors, classroom, school corridor and schoolyard  
(Entire survey period)

	NO <sub>2</sub> value for outdoors	NO <sub>2</sub> value for classroom	NO <sub>2</sub> value for school corridor	NO <sub>2</sub> value for schoolyard
NO <sub>2</sub> value for outdoors	1.00	0.507	0.801	0.989
NO <sub>2</sub> value for classroom		1.00	0.880	0.548
NO <sub>2</sub> value for school corridor			1.00	0.814
NO <sub>2</sub> value for schoolyard				1.00

No. of subjects : 39

Table 2-(2) Correlation coefficient between NO<sub>2</sub> value for outdoors, school room, school corridor and schoolyard  
(Non-heating period)

	NO <sub>2</sub> value for outdoors	NO <sub>2</sub> value for classroom	NO <sub>2</sub> value for school corridor	NO <sub>2</sub> value for schoolyard
NO <sub>2</sub> value for outdoors	1.00	0.861	0.972	0.995
NO <sub>2</sub> value for classroom		1.00	0.962	0.893
NO <sub>2</sub> value for school corridor			1.00	0.938
NO <sub>2</sub> value for schoolyard				1.00

No. of subjects : 14

Table 2-(3) Correlation coefficient between NO<sub>2</sub> value for outdoors, classroom, school corridor and schoolyard  
(Heating period)

	NO <sub>2</sub> value for outdoors	NO <sub>2</sub> value for classroom	NO <sub>2</sub> value for school corridor	NO <sub>2</sub> value for schoolyard
NO <sub>2</sub> value for outdoors	1.00	0.231	0.658	0.986
NO <sub>2</sub> value for classroom		1.00	0.821	0.260
NO <sub>2</sub> value for school corridor			1.00	0.938
NO <sub>2</sub> value for schoolyard				1.00

No. of subjects : 25

Table 3 Arithmetic mean (standard deviation) of NO<sub>2</sub> personal exposure values by month

(ppb)

Year of survey	Survey area	Month					
		Sep.	Oct.	Nov.	Dec.	Jan.	Feb.
1985	A	-	25 (6)	21 (6)	33 (17)	45 (23)	47 (23)
	B	-	12 (4)	14 (14)	32 (25)	34 (24)	35 (21)
1986	A	18 (4)	24 (6)	22 (8)	25 (15)	31 (21)	36 (21)
	C	13 (3)	16 (4)	23 (10)	30 (17)	36 (19)	38 (21)
1987	D	6 (2)	12 (4)	25 (7)	30 (26)	47 (22)	50 (27)
	E	25 (5)	14 (3)	22 (9)	33 (15)	37 (19)	50 (23)

(ppb)

Year of survey	Survey area	Month				
		Mar.	Apr.	May	June	July
1987	C	38 (17)	21 (9)	15 (4)	12 (4)	7 (3)

2023380447

Table 4 Arithmetic mean of NO<sub>2</sub> personal exposure by family smokers and type of heating apparatus

(μg/m<sup>3</sup>/day)

Area	Type of heating apparatus	Family smokers (+)	Family smokers (-)	Total
A	Total	64±38	56±32	59±34
	With ventiduct	47±18	44±17	45±18
	Without ventiduct	90±45	74±39	80±42
B	Total	40±33	46±40	44±38
	With ventiduct	28±33	30±24	29±28
	Without ventiduct	50±30	62±46	57±41
C	Total	46±28	43±30	44±29
	With ventiduct	30±11	29±11	30±11
	Without ventiduct	71±30	70±35	70±33
D	Total	49±44	48±41	48±41
	With ventiduct	30±19	30±22	30±21
	Without ventiduct	91±52	83±47	84±48
E	Total	51±29	52±33	52±32
	With ventiduct	38±14	38±18	38±17
	Without ventiduct	72±34	77±39	75±38

Mean ± S.D.

Table 5 Arithmetic mean of NO<sub>2</sub> personal exposure by type of heating apparatus and housing structure

(μg/m<sup>3</sup>/day)

Area	Type of heating apparatus	Wooden house with wooden sash	Wooden house with aluminium sash	Reinforced concrete house	Total
A	Total	58±32	59±33	58±33	58±34
	With ventiduct	46±18	43±17	46±18	45±18
	Without ventiduct	76±37	79±38	82±49	80±42
B	Total	34±20	44±36	46±40	43±38
	With ventiduct	25±18	24±18	34±35	29±27
	Without ventiduct	44±21	59±40	61±49	58±41
C	Total	41±25	45±30	47±30	44±29
	With ventiduct	30±11	29±11	33±15	30±11
	Without ventiduct	58±29	72±34	81±31	70±33
D	Total	50±41	48±43	47±39	48±41
	With ventiduct	28±17	30±23	32±20	30±21
	Without ventiduct	83±45	84±49	87±47	80±48
E	Total	49±29	54±35	51±29	52±32
	With ventiduct	35±14	36±16	42±18	38±17
	Without ventiduct	71±34	77±39	76±36	75±38

Mean ± S.D.

2023380448

significant. However, for the heating period, the regression coefficients of the use of heating apparatus without ventiduct, ambient NO<sub>2</sub> level and housing structure factors were of significance. The regression coefficient of outdoor NO<sub>2</sub> level was notably 0.417 for the non-heating period and 0.395 for the heating period.

Then an investigation was performed to determine the propriety of the appraisal of NO<sub>2</sub> exposure levels in the epidemiological study, i.e. the estimation of NO<sub>2</sub> personal exposure levels on a group level.

Fig. 3 represents the relationship between the mean NO<sub>2</sub> exposure levels (group NO<sub>2</sub> exposure levels) of all subjects in each survey and the ambient NO<sub>2</sub> level monitored at that time using the filter badge placed in the monitoring station. The open circles on the chart represent NO<sub>2</sub> values in the non-heating period and the black circles NO<sub>2</sub> values in the heating period. The NO<sub>2</sub> group exposure levels for the non-heating period as represented by the open circles and the NO<sub>2</sub> levels measured at the monitoring station were judged to show a linear relationship. In addition, excluding the one stray point, a linear relation was also judged present for the heating period. A multiple regression analysis was performed on this data, the results of which are shown in Table 7.

These results suggest that NO<sub>2</sub> group exposure levels in the non-heating period may be explained by ambient NO<sub>2</sub> levels and in the heating period by ambient NO<sub>2</sub> level and percentage use of non-ventilated heating apparatus. The regression coefficient of the ambient NO<sub>2</sub> level was 0.431 for the non-heating period, 0.398 for the heating period and 0.403 for the entire survey period. Accordingly, it was judged feasible to use the predicted equations shown in the bottom column of the table for estimating NO<sub>2</sub> personal exposure levels as on a group level.

We obtained already a significant negative correlation coefficient (0.79) between the percentage use of heating apparatus and 24-hour mean temperature. Therefore, it should still be possible to estimate NO<sub>2</sub> personal exposure levels on a group level even if we substituted non-ventiduct heating apparatus percentage use with mean temperature (see Table 8). In other words, we believe it is possible to estimate mean NO<sub>2</sub> exposure levels per day per person in the group without examining the non-ventiduct heating apparatus percentage use as long as you can measure the ambient NO<sub>2</sub> level and mean temperature. At least we feel this applies to the estimation of the daily NO<sub>2</sub> exposure level on a group level for the entire survey period and the heating period.

In order to ensure the validity of our predicted equation shown in Table 7, we examined measurement data collected once a month (excluding August) for 11 months from October 1983 to September 1984, using as subjects 80 schoolchildren in Yao City, Osaka Prefecture. The results of this study are shown in Fig. 4. The correlation coefficient between the observed and calculated values is very high at 0.977, and it could be said that observed and calculated values of NO<sub>2</sub> exposure levels are almost identical. Therefore, it appears possible that the NO<sub>2</sub> exposure level obtained by the predicted equations derived in this research study can be used for estimating NO<sub>2</sub> personal exposure levels in the epidemiological study.

2023380419

Table 6 Results of multiple regression analysis between NO<sub>2</sub> personal exposures and various factors

		Non-heating period	Heating period	Entire survey period
Regression coefficient	NO <sub>2</sub> value for outdoors	0.417**	0.395**	0.445**
	Type of heating apparatus	-	17.303**	19.725**
	Housing structure	0.437	3.789**	2.667
	Cookery gas apparatus	-0.296	0.168	0.130
	Presence of family smokers	0.733**	0.629	0.497
Constant		7.1	9.5	6.7
Number of subjects		1.910	3.473	5.383
Multiple regression coefficient		0.749**	0.468**	0.611**

- NO<sub>2</sub> personal exposure value : ppb
  - NO<sub>2</sub> value for outdoors : ppb. average/day (by NO<sub>2</sub>-filter badge method)
  - Type of heating apparatus : (heating apparatus with ventiduct: 0, without ventiduct: 1)
  - Housing structure : (wooden house with aluminium sash : 0, the others : 1)
  - Cookery gas apparatus : (not used : 0, used: 1)
  - Presence of family smokers : (yes:0, no : 1)
- \*\* P<0.01

Table 7 Result of multiple regression analysis between mean of NO<sub>2</sub> personal exposure values as on a group level and various factors

		Non-heating period	Heating period	Entire survey period
Regression coefficient	NO <sub>2</sub> value for outdoors	0.431**	0.398**	0.403**
	Items of heating	-	0.316**	0.294**
Constant		7.6	6.4	7.7
Number of subjects		14	25	39
Multiple regression coefficient		0.926**	0.887**	0.949**
A predicted equation to estimate NO <sub>2</sub> personal exposure levels		Y=0.431a+7.6	Y=0.398a+0.316b+6.4	Y=0.403a+0.294b+7.7

- Mean of NO<sub>2</sub> personal exposure values as on a group : ppb (average/day)
  - NO<sub>2</sub> value for outdoors : NO<sub>2</sub> value measured at monitoring station, ppb.(average/day)
  - Items of heating apparatus : Number of heating apparatus used / number of surveys × 100(%)
- \*\* P<0.01

Table 8-(1) Results of multiple regression analysis (NO<sub>2</sub> personal exposure value, outdoor NO<sub>2</sub> value, air temperature) (Entire survey period)

Factors	Partial correlation coefficient	Regression coefficient	Standardized partial correlation coefficient	t-value
NO <sub>2</sub> value for outdoors	0.618	0.363	0.369	4.72**
Mean value for air temperature	-0.835	-1.137	-0.712	9.11**

N=39

Multiple correlation coefficient: 0.894  
 $Y = 0.363a - 1.137c + 31.9$

\*\* P&lt;0.01

Table 8-(2) Results of multiple regression analysis (NO<sub>2</sub> personal exposure value, outdoor NO<sub>2</sub> value, air temperature) (Heating period)

Factors	Partial correlation coefficient	Regression coefficient	Standardized partial correlation coefficient	t-value
NO <sub>2</sub> value for outdoors	0.576	0.373	0.420	-3.31**
Mean value for air temperature	-0.755	-1.789	-0.685	5.40**

N=25

Multiple correlation coefficient: 0.804  
 $Y = 0.373a - 1.789c + 36.3$

\*\* P&lt;0.01

2023380450

However, it must be borne in mind that the results of this survey were obtained using as subjects schoolchildren whose daily behavioral pattern within the housing structure was similar and who experienced only Osaka climate. In areas with a colder climate than in Osaka, the heating period, method and housing structure will differ and even in the Osaka region age ranges outside that of the children tested would have differing daily life patterns and thus it would not be possible to use the equation directly resulting from this study in their original form. In addition, the object area for this study did not face onto a main road. Therefore, it wouldn't be appropriate to apply these equations in measuring the NO<sub>2</sub> personal exposure levels of schoolchildren living in the vicinity of a main road.

#### REFERENCES

(Omitted)

2023380451

# **PRESENT AND FUTURE OF INDOOR AIR QUALITY**

**Proceedings of the Brussels Conference,  
14–16 February 1989**

*Editors:*

**C.J. Bieva, Y. Courtois and M. Govaerts**



1989

**EXCERPTA MEDICA, Amsterdam – New York – Oxford**

2023380452

© 1989 Elsevier Science Publishers B.V. (Biomedical Division)

All rights reserved. No part of this publication may be reproduced, stored in a retrieval system or transmitted in any form or by any means, electronic, mechanical, photocopying, recording or otherwise without the prior written permission of the publisher, Elsevier Science Publishers B.V., Biomedical Division, P.O. Box 1527, 1000 BM Amsterdam, The Netherlands.

No responsibility is assumed by the Publisher for any injury and/or damage to persons or property as a matter of products liability, negligence or otherwise, or from any use or operation of any methods, products, instructions or ideas contained in the material herein. Because of rapid advances in the medical sciences, the Publisher recommends that independent verification of diagnoses and drug dosages should be made.

Special regulations for readers in the USA - This publication has been registered with the Copyright Clearance Center Inc. (CCC), 27 Congress Street, Salem, MA 01970, USA. Information can be obtained from the CCC about conditions under which photocopies of parts of this publication may be made in the USA. All other copyright questions, including photocopying outside the USA, should be referred to the copyright owner, Elsevier Science Publishers B.V., unless otherwise specified.

International Congress Series No. 860  
ISBN 0 444 81134 6

*This book is printed on acid-free paper.*

*Published by:*  
Elsevier Science Publishers B.V.  
(Biomedical Division)  
P.O. Box 211  
1000 AE Amsterdam  
The Netherlands

*Sole distributors for the USA and Canada:*  
Elsevier Science Publishing Company Inc.  
655 Avenue of the Americas  
New York, NY 10010  
USA

Printed in The Netherlands.

2023386453

## FOREWORD

Indoor air quality conservation and procedures for the measurement of related potential pollutants, such as radon, asbestos, gases, pesticides, tobacco smoke and bacteria from air conditioning systems, have seen important changes in recent years, while the range and the scope of the studies have continued to expand.

In addition to helping preserve public health, the field of interest is now extending to include such areas as architectural design, ventilation engineering, sociology, psychology and legal aspects. Related analytical techniques like gas chromatography and mass spectroscopy have undergone parallel refinements and their range of application has broadened.

These advances were discussed at the Conference 'Present and Future of Indoor Air Quality', held in Brussels, February, 1989, following symposia on indoor air quality at Essen and Tokyo in 1987 and London in 1988. The sessions were attended by about 200 scientists representing 20 countries. A total of 92 papers and posters were presented covering such topics as pathogenesis and epidemiology, sources of indoor air contamination and risk assessment, chemistry of indoor air related to the outdoor air quality, social and psychological aspects of poor indoor air quality, motivation and attitudes, future guidelines for the improvement of indoor air quality through architectural and ventilation design, and air quality monitoring.

The proceedings include full texts and posters presented during the meeting. The organising committee hopes that they will constitute a useful guide for the improvement of our indoor air quality in the future.

2023380454

# CONTENTS

## **OPENING SESSION: RISK ASSESSMENT AND HEALTH**

Cancer control and lifestyle medicine <i>E.L. Wynder</i>	3
Sick building syndrome – Clinical aspects and prevention <i>C. Molina</i>	15
The ambient air, perceptions and tolerance thresholds: Changing social standards <i>N. Delruele</i>	23
Concerns and risks of home pollution assessed from the 329 cases reported to a Poison Control Centre during 1987 <i>G. Riboulet-Delmas, R. Garnier and M.-L. Efthymiou</i>	29
Carbon monoxide poisoning: Epidemiology and prevention <i>B. Tissot, M. Govaerts and N. Tinant</i>	37
Current state of research on air quality inside buildings <i>J.C. Loewenstein</i>	43
Radon and lung cancer: A case-control study in southern Belgium <i>A. Poffijn, R. Mak, P. Weynants, M. Vanhoorne and J. Prignot</i>	51
Indoor radon in Belgium <i>A. Poffijn and H. Vanmarcke</i>	55

## **SECTION 1: ETS AND PASSIVE SMOKING**

Environmental tobacco smoke and lung cancer: Is it the smoke or the diet? <i>L.C. Koo</i>	65
Dietary habits are of limited importance in influencing the lung cancer risk among Japanese females who never smoked <i>T. Hirayama</i>	77
An example of extra-poisson variation suggesting an under-specified model <i>S.J. Kilpatrick</i>	83
Lung cancer risk by oral exposure <i>R. Rylander, H.-J. Haussmann and F.J. Tewes</i>	91
Passive smoking: Acute effects on lung function of sensitive persons <i>B. Danuser, A. Weber and A.L. Hartmann</i>	101
Evaluation of exposure to ambient tobacco smoke: A few concrete examples concerning the value of biological and atmospheric indicators <i>A.M. Laurent, Y.A. Courtois, S. Pretet, J.R. Claude and B. Festy</i>	103

2023380455

Exposure to environmental tobacco smoke in the non industrial workplace under different conditions of ventilation and smoking regulation.	
<i>T. Sterling, C. Collett and J. Ross</i>	111
Passive smoking and lung cancer; fact or fiction?	
<i>P. Lee</i>	119
Initiatives taken within parents in school to reduce passive smoking risks	
<i>C. Arciti, B. Doglio and L. Santi</i>	129
Health care and society: Environmental tobacco smoke and lung cancer	
<i>E.L. Wynder and G.C. Kabat</i>	135
Isoprene - A potential indoor indicator for environmental tobacco smoke.	
<i>G. Löfroth</i>	147
The passive smoking myth	
<i>N. Mantel</i>	155
Relative lung cancer risk from exposure to mainstream and sidestream smoke particulates	
<i>J.J. McAughey, J.N. Pritchard and A. Black</i>	161
Measurement of environmental tobacco smoke in an air-conditioned office building	
<i>C.J. Proctor, N.D. Warren and M.A.J. Bevan</i>	169
Nicotine and indoor air pollution	
<i>C. Arfi, J. Kaloustian, A.M. Pauli, J. Pastor, F. Grimaldi, F. Gouezo and A. Viala</i>	173
Assessing the validity of a Japanese cohort study	
<i>M.W. Layard and J.R. Viren</i>	177
<b>SECTION 2: INDOOR AIR QUALITY: CHEMISTRY AND BIOLOGICAL EFFECTS</b>	
Exposure to ETS and its biological effects: A review	
<i>F.X. Adlkofer, G. Scherer, L. Von Meyerinck, Ch. Von Maltzan and L. Jarczyk</i>	189
Carbonyl compounds in mainstream and sidestream cigarette smoke	
<i>H. Schlitt and H. Knöppel</i>	197
Sensory responses to environmental tobacco smoke from cigarettes that heat but do not burn tobacco	
<i>J.C. Walker, R.A. Jennings, W.T. Morgan, J.H. Robinson, D.W. Griffith and J.H. Reynolds IV</i>	207
Role of combustion products in bronchial hyperreactivity - A case control study	
<i>M. Bert, J. Kühr, A. Hendel-Kramer, T. Urbanek, E. Schultz, H. Steiger and W. Karmaus</i>	215
The importance of endotoxin and glucan for symptoms in sick buildings	
<i>R. Rylander, S. Sørensen, H. Goto, K. Yuasa and S. Tanaka</i>	219

2023380456

An epidemiological study on dose-response relationship between B(a)P concentrations in indoor air and lung cancer mortality in Xuan Wei, China <i>He Xingzhou, Chen Wei and Chen Hong</i>	227
Health consequences of atopy, food intolerance and indoor environment: A case study <i>E. Sammaljärvi</i>	235
Assessment of exposure to emissions from a liquefied petroleum gas stove used in urban India <i>V. Haraprasad and J.M. Dave</i>	239
Quality of indoor environment in dwellings according to performance of heating system <i>D. Petráš</i>	243
Oxides of nitrogen and respiratory illness. Is there a susceptible population? <i>J.M. Sneddon and T.M. Bearpark</i>	247
The effect of indoor air pollution on health from coal burning containing high fluorides <i>Ji Rongdi and Cao Shouren</i>	253
Effect of reservoir fuel levels on emissions from a kerosene cook stove <i>J.M. Dave and V. Haraprasad</i>	257
Study of the small-scale relations between indoor and outdoor pollution (SO <sub>2</sub> - NO <sub>x</sub> - particles) in a hospital in the Paris region <i>J.C. Loewenstein</i>	261
Nicotine absorption in humans following exposure to environmental tobacco smoke generated from different types of cigarettes <i>J.D. deBethizy, L.E. Bates, R.A. Davis, D.L. Heavner, P.R. Nelson, J.C. Walker and J.H. Robinson</i>	269
Characterization of the environmental tobacco smoke generated by different cigarettes <i>P.R. Nelson, D.L. Heavner and B.B. Collie</i>	277
Health and social problems of carbon monoxide poisoning from the usage of anthracite coal in Korea <i>Yong Chung, Dongchun Shin and Youngwook Lim</i>	283
On the use of environmental tobacco smoke component ratios <i>G.B. Oldaker III, W.E. Crouse and R.M. Depinto</i>	287

### SECTION 3: INDOOR AIR QUALITY: VENTILATION, CLIMATISATION AND POLLUTANTS

Ventilation, health and energy conservation - A workable compromise <i>G. Robertson</i>	293
Ventilation system retrofits as a method of solution for the 'sick building syndrome' <i>C. Collett and E. Sterling</i>	301

2023380457

A few studies of air-conditioned environments <i>A. Mouilleseaux, F. Squinazi and B. Festy</i>	309
Regression model for indoor concentrations of combustion-generated gases <i>A. Hendel-Kramer, J. Kühr, R. Urbanek, H. Staiger, E. Schultz and E. Karmaus</i>	315
A Czechoslovak survey about ventilation habits in dwellings <i>L. Piršel</i>	321
Emissions from materials; The role of additives in PVC <i>K. Saarela, K. Kaustia and A. Kiviranta</i>	329
Influence of indoor air pollution on NO <sub>2</sub> personal exposure levels of schoolchildren <i>Y. Shimizu, S. Imai and Y. Tsunetoshi</i>	337
Indoor pollution by mineral fibers <i>J. Bignon, G. Dufour, M.A. Billon-Galand, A. Gaudichet, H. de Cremoux and P. Brochard</i>	347
The Acarex/Acarosan concept, its possibilities in allergology and dermatology <i>J.E.M.H. Van Bronswijk, G. Schober and F.M. Kniest</i>	355
Elimination of house dust mite excreta, the carriers of allergens in the indoor air <i>E. Bischoff, A. Fischer and B. Liebenberg</i>	363
Portable air sampler for measurements in aircraft and public buildings <i>J.F. van der Wal</i>	371
Destruction of acarids and biodecontamination of domestic environment <i>M. Blanc and Ch. Boutin</i>	379
Dust in the work environment: Effect of pharmacological agents on the airway response to inhaled cotton dust <i>P.J. Nicholls, M. McDermott, M. Bevan and D. Griffiths-Johnson</i>	385
Moulds, mites and moisture: A preliminary report on six cases of fungal damage in dwellings <i>H.S.M. Kort, J.E.M.H. Van Bronswijk and G. Schober</i>	389
Absolute indoor air humidity and the abundance of allergen producing house dust mites and fungi in the Netherlands <i>G. Schober</i>	395
Air quality control of public car parks <i>F. Cupelin and J.Cl. Landry</i>	401
<b>SECTION 4: INDOOR AIR QUALITY: PREVENTION AND FUTURE GUIDELINES</b>	
Demand controlled ventilating systems: New concepts for indoor air quality and energy conservation <i>L. Trepte</i>	407
A database of problem buildings: Learning by past mistakes <i>C.W. Collett, E.M. Sterling, T.D. Sterling and J.J. Weinkam</i>	413

2023380458

Treatment programme for microbiologically contaminated airconditioning installations <i>E. Clement and R. De Koster</i>	421
Present and future exceedance of health related reference values for air pollutants in Dutch dwellings <i>H.J. Van de Wiel and E. Lebret</i>	427
French incentive actions for research on indoor air quality in buildings <i>J.-P. Marie, J. Dubois and P.-C. Jacquignon</i>	435
A nitrogen oxide analyser designed for the study of indoor pollution: The APPI-AC35 <i>B. Millancourt</i>	443
Automatic analyser prototype for atmospheric isoprene and monoterpenes <i>M.L. Riba, B. Clement, M. Haziza and L. Torres</i>	449
Air tightness, permeability or transparency to air in buildings, different names for one same problem <i>M. Kilberger</i>	453
<b>Index of authors</b>	461

2023386459

## REGRESSION MODEL FOR INDOOR CONCENTRATIONS OF COMBUSTION-GENERATED GASES

A. Hendel-Kramer, J. Kühr, R. Urbanek,  
H. Staiger\*, E. Schultz\* & W. Karmaus\*\*

(University Children Hospital, Freiburg, \*German Weather  
Service, Freiburg &  
\*\*Science Centre, Berlin, FRG)

NOTICE:  
This material may be  
protected by copyright  
law (Title 17, U.S. Code).

### Introduction

We measured indoor- and outdoor-concentrations of NO<sub>2</sub> and SO<sub>2</sub> within an epidemiological study about the correlation between indoor air pollution and respiratory diseases in childhood. A regression-model for concentrations of combustion-generated gases was set up, which allows estimations about the correlations between the concentrations and other variables.

### Methods

In 116 dwellings in the region of Freiburg and of the Black-Forest the sampling of nitrogen dioxide (NO<sub>2</sub>) and sulphur dioxide (SO<sub>2</sub>) was performed by Palmes diffusion tubes. At the same time outdoor data were collected at 36 stations near the residences. The sampling was carried out over a period of october 87 to may 88. The absorbed gases were analyzed by ionchromatography. The exploration of indoor conditions resulted from a standardized interview with the housewife.

The following data were considered for the regression-model:

#### 1. As dependent variables:

- NO<sub>2</sub> indoor-concentrations
- SO<sub>2</sub> indoor-concentrations

#### 2. As independent variables:

- NO<sub>2</sub> respectively SO<sub>2</sub> outdoor-concentrations
- outdoor air temperature
- tobacco smoking in the dwelling
- kind of stoves
- gas-cooking
- ventilation characteristics of the dwelling

### Description of variables

According to the number of smoked cigarettes four categories for tobacco smoking were established.

2023380460

Tab. 1  
NO<sub>2</sub>-Model (Frequencies of the variables)

Gas cooking in dwelling	number of observations %
yes	12.8
no	87.2
	100.0 (N = 428)

Tobacco smoking in dwelling	number of observations %
yes	25.7
no	74.3
	100.0 (N = 428)

Kind of stoves	number of observations %
central heating	56.5
one stove with fossil fuels	15.0
more than one stove with fossil fuels	18.5
	100.0 (N = 428)

Ventilation classes	number of observations %
class 0 + 1	73.1
class 2 + 3	26.9
	100.0 (N = 428)

2023380461

The dwellings were categorized considering the kinds of fuel and heating.

The ventilation characteristics were registered by the kind of building (e.g. prefabricated house) and by the habits of the housewife to ventilate.

Because the dependent and independent variables have continuous values a linear regression-analysis was used. It requires a normal distribution of the considered values. Therefore we performed a transformation with natural logarithm. The formula  $y = (-1 + e^{PAR} \cdot x) \cdot 100$  gives the percentage increase of the dependent variable if the values of the independent variables are increasing.

#### Results

#### NO<sub>2</sub>-MODEL

(Tab.1 ; Fig.1)

N = 428 values for the NO<sub>2</sub> indoor-concentrations are available.

The range of the values is 2 µg/m<sup>3</sup> to 201 µg/m<sup>3</sup> indoors and 3 µg/m<sup>3</sup> to 116 µg/m<sup>3</sup> outdoors.

The results of the regression analysis are:

Variables	Regr.-Coefficient	Significance-Level
Intercept	2.613	0.0001
NO <sub>2</sub> outdoor conc.	0.005	0.0047
Outdoor temperature	0.030	0.0003
Gas cooking	0.327	0.0044
Ventilation charact.	0.051	0.0005
Smoking	- 0.026	0.2904
Kind of stoves	0.041	0.1299

The model predicts the following connections:

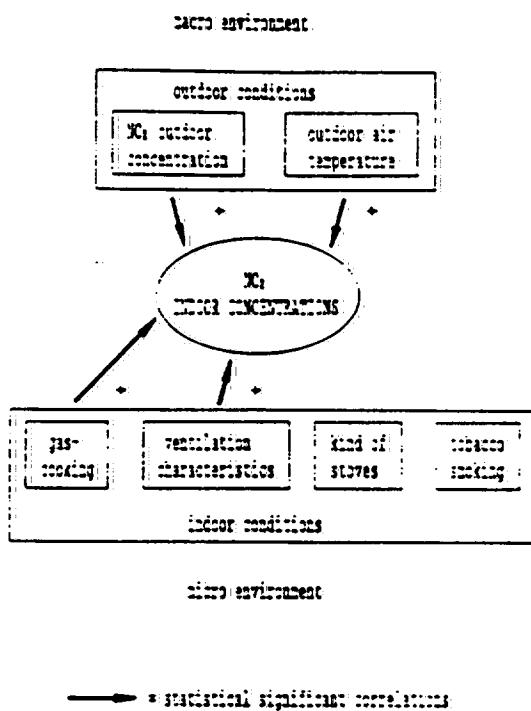
1. The higher the NO<sub>2</sub> outdoor-concentrations the higher the indoor-concentrations.
2. The higher the outdoor air temperature the higher the NO<sub>2</sub>-indoor-concentrations.

An explanation for this is the ventilation habit. Opening of the windows at higher temperature causes an increase in indoor-concentrations.

3. In dwellings with gas-cooking the NO<sub>2</sub> concentrations are higher than in others.
4. In well isolated dwellings with low ventilation rates the NO<sub>2</sub> concentrations are higher than in others.

2023380462

Fig. 1

DETERMINANTS OF NO<sub>2</sub> INDOOR CONCENTRATIONS

These four variables are statistically significant. Tobacco smoking and the kind of stoves have no significant effect on the NO<sub>2</sub> indoor-concentrations in our model.

This model predicts 8% of the variance.

SO<sub>2</sub>-MODEL

(Tab.2 ; Fig.2)

N = 262 values are the basis of the computation. The range of the values goes from 1 µg/m<sup>3</sup> - 136 µg/m<sup>3</sup> indoor and 1 µg/m<sup>3</sup> to 163 µg/m<sup>3</sup> outdoor.

The results of the regression analysis are:

Variables	Regr.-Coefficient	Significance-Level
Intercept	2.010	0.0001
SO <sub>2</sub> outdoor conc.	0.014	0.0001
Outdoor temperature	- 0.095	0.0001
Gas cooking	0.457	0.0188
Ventilation charact.	0.051	0.5662
Smoking	- 0.005	0.8942
Kind of Stoves	- 0.034	0.4778

2023380463

Tab. 2

## Model (Frequencies of variables)

Gas cooking in dwelling	number of observations %
yes	13.0
no	87.0
	100.0 (N = 262)

Tobacco smoking in dwelling	number of observations %
yes	24.8
no	75.2
	100.0 (N = 262)

Kind of stoves	number of observations %
central heating	66.8
one stove with fossil fuels	17.2
more than one stove with fossil fuels	16.0
	100.0 (N = 262)

Ventilation classes	number of observations %
class 0 + 1	72.9
class 2 + 3	27.1
	100.0 (N = 262)

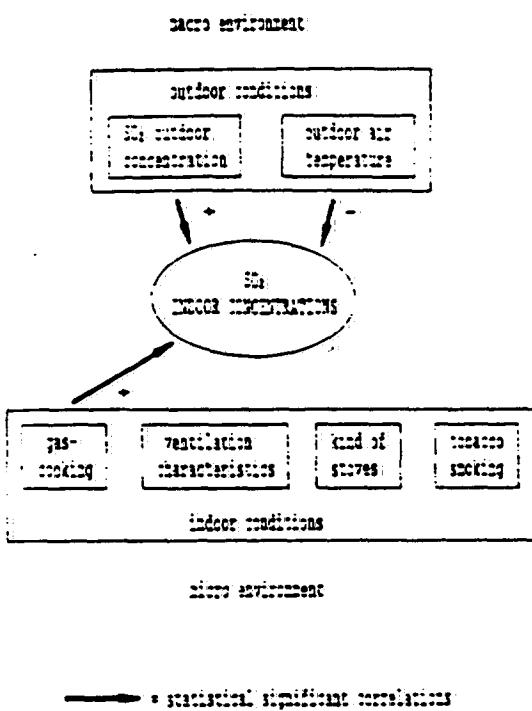
Three of the checked 6 variables are statistically significant.

The model predicts the following connections:

1. The higher the SO<sub>2</sub> outdoor-concentrations, the higher the indoor-concentration.
2. The higher the outdoor air temperature the lower the SO<sub>2</sub> indoor-concentrations. An explanation for this connection is the lower heating activities in the warm season, which causes reduced SO<sub>2</sub> outdoor-concentrations.

2023380464

Fig. 2

DETERMINANTS OF SO<sub>2</sub> INDOOR CONCENTRATIONS

3. In dwellings with gas cooking the SO<sub>2</sub> indoor-concentrations are higher than in other. Because gas cooking is no source of SO<sub>2</sub> we assume, that the ventilation behavior is responsible for this connection.

This model predicts 23,3% of the variance.

#### Summary

Our epidemiological data are obtained under "field conditions". Concerning the indoor-concentration of NO<sub>2</sub> the statistically significant variables are NO<sub>2</sub> outdoor-concentration, outdoor air temperature, gas cooking and ventilation characteristics. Concerning the indoor-concentration of SO<sub>2</sub> the statistically significant variables are SO<sub>2</sub> outdoor concentration, outdoor air temperature and gas cooking.

Our models only give a small explication percentage for the variance. Therefore the indoor gas concentrations couldn't be predicted with potential determinants of the indoor and outdoor environment. For epidemiological studies the actually registered data in analyzed dwellings must be considered.

2023380465

008281

T 87/11/3/12

# Thermal Desorption/Gas Chromatographic/Mass Spectrometric Analysis of Volatile Organic Compounds in the Offices of Smokers and Nonsmokers

EM/USA

Charlene W. Bayert and Marilyn S. Black

Georgia Tech Research Institute, Analytical and Instrumentation Branch, Emerson A112, Atlanta, Georgia 30332, USA

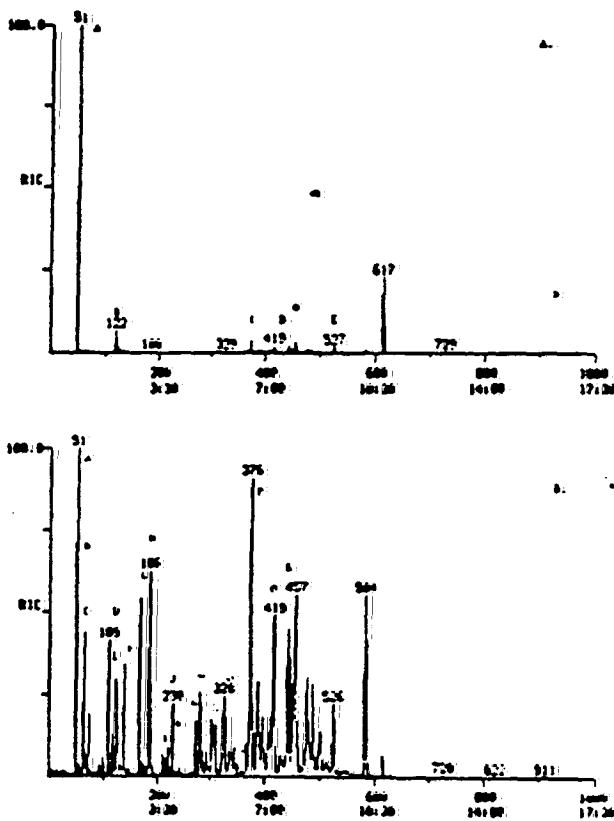
The indoor air quality of the offices of smokers and nonsmokers was surveyed for volatile organic compound identities and concentrations. These results were examined to determine whether environmental tobacco smoke contamination could be distinguished from airborne pollutants outgassing from other sources. It was not possible to positively attribute volatile organic contaminants to environmental tobacco smoke. It was possible to distinguish between smokers' and nonsmokers' offices by determining airborne nicotine levels.

## INTRODUCTION

Indoor air quality has recently become a major issue and research area. The concentrations of many airborne contaminants are significantly greater inside buildings and residences where the average person spends approximately 90 per cent of his/her time<sup>1,2</sup> (Fig. 1). A multitude of sources can yield potentially harmful vapors such as tobacco smoke, building materials and furnishings, copy machines, cooking and heating fuels, aerosol propellants, cleaning compounds, dry cleaning solvents, printed paper, etc.<sup>3-5</sup> The sick-building syndrome<sup>6</sup> can result in a number of nonspecific symptoms. These include headaches, eye irritation, fatigue, dry throat, sinus congestion, dizziness, and nausea. Sick-building syndrome is difficult to measure since it usually is caused by an interplay of many substances as well as how the indoor environment is controlled. Volatile organic compounds (VOCs) are one class of pollutants which have come under particular scrutiny. These substances are ubiquitous in the indoor environment and comprise a very complex matrix.

Capillary gas chromatography/mass spectrometry (GC/MS) is the preferred technique for detection and identification of the broad spectrum of VOCs in survey air samples. Three sample introduction modes are employed: (1) direct thermal desorption of gases collected on solid sorbents<sup>7-11</sup> (2) liquid injection of solvent extract of gases collected on solid sorbents or filters<sup>12</sup> (3) direct gaseous injection of grab samples.<sup>13</sup> Solid sorbent collection with subsequent thermal desorption GC/MS (TD/GC/MS) analysis offers significant advantages including: selective adsorption of organic species, effective preconcentration of organic species on solid sorbent during sample collection, and direct thermal injection with minimal sample handling and preparation. Sensitivity, accuracy, and precision of the overall analysis technique are enhanced by solid sorbent/TD/GC/MS.

<sup>1</sup> Author to whom correspondence should be addressed.



Environmental tobacco smoke (ETS) is one of the most controversial sources of indoor air contaminants. ETS is that portion of smoke to which the passive smoker is exposed. It is defined as a combination of the smoke emanating from the burning end of an idling cigarette or cigar (sidestream smoke) and exhaled smoke (exhaled mainstream smoke). ETS exposure is difficult to establish due to: (1) the complexity of smoke composition; (2) the similarity of ETS constituents to those outgassed from other volatile pollutant sources; and (3) the variability of ETS composition. Over 3000 compounds, including volatile organic and inorganic species, have been identified in tobacco smoke.<sup>14</sup> Numerous nitrogenous compounds have been identified in mainstream smoke.<sup>15-21</sup> These compounds are not commonly detected in indoor air so the presence of nitrogenous compounds may serve as markers for ETS contamination.

An investigation was undertaken to survey and compare the indoor air quality in offices of smokers and nonsmokers. Most previous studies to date have used respirable particles<sup>22</sup> or nicotine<sup>23</sup> as a measure of ETS levels. This study examined the presence and concentrations of VOCs and nicotine. It was hoped to be able to find correlations between VOC identities and concentrations and the presence of ETS.

## EXPERIMENTAL

### Solid sorbent tubes

Tenax GC 60/80 mesh (Alltech Associates) was Soxhlet extracted for 24 h each with methanol and hexane. The adsorbent was dried under a nitrogen stream prior to packing into a clean glass tube. Two hundred mg were packed into a clean glass tube (4 mm i.d.  $\times$  180 mm length) plugging the tube ends with glass wool. The tubes were conditioned at 270 °C for a minimum of 18 h, while being flushed with a stream of 20 ml min<sup>-1</sup> nitrogen. Tubes were cooled to room temperature while continuing to flush with nitrogen, then placed in clean aluminum, o-ring sealed containers (Tekmar, Inc.) for transportation to and from sampling sites. Orbo-42 tubes (Supelco) were used as supplied by manufacturer.

### Sampling and desorption

VOCs were sampled during field surveys of office buildings and during experimental studies with a small scale chamber (Fig. 2) attached to a smoking machine. Personal sampling pumps (Dupont 4000) were used to draw air at 100 ml min<sup>-1</sup> for 4 h through the prepared Tenax sorbent tubes at each sampling site. After sampling was complete, the tubes were stored in the aluminum containers at 0 °C until analysis by TD/GC/MS.

Nicotine sampling was conducted in office buildings on Orbo-42 solid sorbent tubes. Personal sampling pumps (Gilian Model 113A) were used to draw air at 1 LPM for 2 h through the sorbent tubes. Front and back portions of tubes were analysed separately to monitor possible breakthrough during sampling. Nicotine was

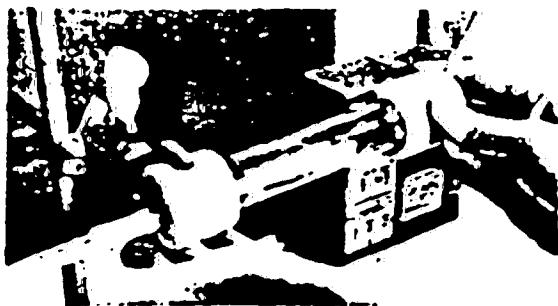


Figure 2. Sidestream smoke collection chamber.

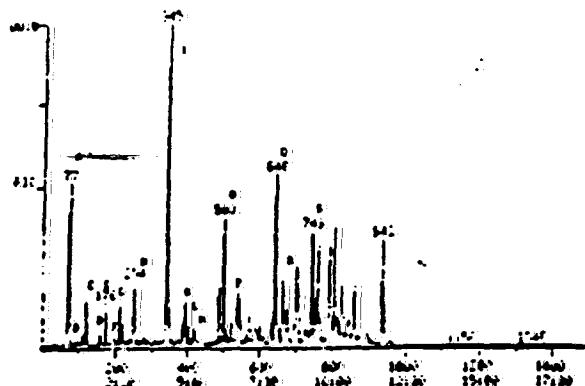
desorbed by extracting the adsorbent with 1 ml ethyl acetate (Baker Resianalysed) for 30 min.

### Instrumentation

Mass spectrometric measurements were obtained with a Finnigan OWA Model 30B GC/MS operating under electron impact conditions (70 eV, 100 mA). Thermal desorption of VOCs preconcentrated on Tenax was accomplished with a Tekmar Model 5000 thermal desorber interfaced with the GC/MS. Helium was used as carrier gas for both the desorber and the GC and was adjusted so that the carrier flow through the capillary GC column was 1.6 ml min<sup>-1</sup>. The desorber was equipped with a cryofocusing unit to trap volatiles on a length of deactivated fused silica (0.32 mm  $\times$  300 mm). The fused silica trap was attached to the capillary column using a butt-end connector (Alltech Associates). The following conditions were used for thermal desorption: purging of tube for 5 min at 45 °C; desorption at 285 °C for 8 min; cooling cryotrap at -150 °C with liquid nitrogen during desorption and transfer through the transfer line; transfer temperature of 285 °C for 1.75 min; valve temperature of 285 °C; injection temperature of 220 °C; and an injection time of 1.5 min. The gas chromatograph separation was begun immediately following flash evaporation from the fused silica trap onto the head of the capillary GC column. A bonded SE54 column (25 m  $\times$  0.32 mm i.d. coated and a 0.5 µm film thickness) (Hewlett-Packard) was used with the following temperature program: 30 °C isothermal for 1 min; 8 °C min<sup>-1</sup> to 250 °C; isothermal for 10 min; and GC/MS transfer over 275 °C. Mass spectral data were collected between 42-500 u in 0.7 sec. External standards were prepared by injecting a known concentration of the analyte of interest in methanol onto a blank solid sorbent tube. This was analysed under identical conditions as the sample tubes.

Nicotine analyses were carried out on a Hewlett-Packard Model 5890 gas chromatograph equipped with a nitrogen/phosphorous detector (NPD). Samples were injected on a DB5 column (30 m  $\times$  0.25 mm i.d. and 0.25 µm film thickness) (J & W Scientific). The following chromatographic isothermal conditions were used: oven temperature 120 °C; injection temperature 260 °C; and detector temperature 260 °C. The detector gas flows were hydrogen 3.0 ml min<sup>-1</sup>, helium 30 ml min<sup>-1</sup>, and air 80 ml min<sup>-1</sup>. Helium was used as the carrier gas at a flow of 1.6 ml min<sup>-1</sup>.

2023380463



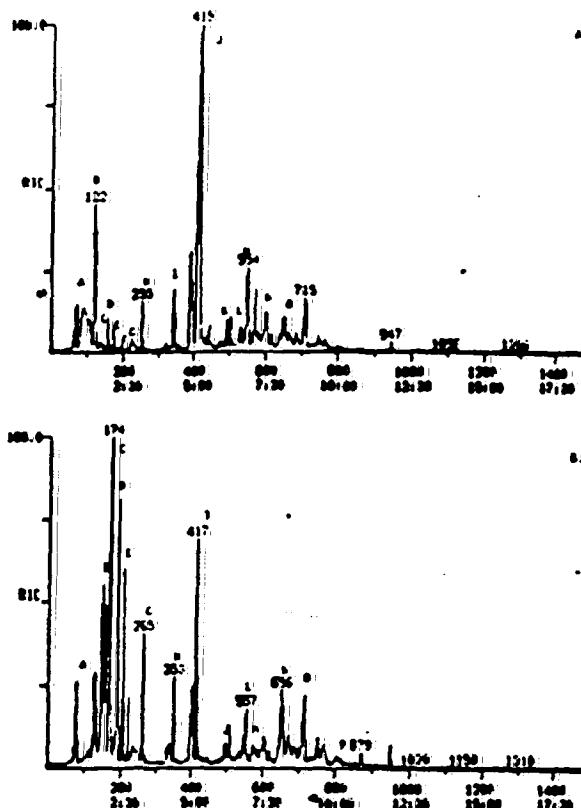
**Figure 3.** Reconstructed ion chromatogram of VOCs detected in an office building. Collection on Tenax; analysis by TD/GC/MS. Peak identifications: A, carbon dioxide; B, isopropanol; C, methylene chloride; D, hexane; E, ethyl acetate; F, 1,1,1-trichloroethane; G, benzene; H, trichloroethylene; I, toluene; J, methylcyclohexane; K, acetone; L, tetrachloroethylene; M, butyl acetate; N, ethylbenzene; O, o-xylene; P, m-xylene; Q, dimethyldecane; R, ethylmethylbenzene; S, dimethylundecane.

## RESULTS AND DISCUSSION

The complexity of the VOC composition typically detected in the indoor atmosphere is depicted in Fig. 3. Building materials and furnishings are the most common sources of these VOCs. Table 1 lists several of the most often detected substances. This VOC building background makes it difficult to distinguish ETS contamination originating from the offices of smokers from the VOCs outgassing from other sources. Figs 4 and 5 show reconstructed ion chromatograms obtained by air sampling on Tenax in two different office buildings. Each figure compares the ambient VOCs in the office of a smoker versus the office of a nonsmoker. Only minor differences are detected between the two areas. The

**Table 1.** Common VOCs detected in indoor air

Aliphatic Hydrocarbons:	
Hexane	
Decane	
Undecane	
Methylcyclohexanes	
Dimethylheptanes	
Alkylated Aromatic Hydrocarbons:	
Toluene	
Xylenes	
Ethylbenzene	
Methylnaphthalenes	
Chlorinated Hydrocarbons:	
Tetrachloroethylene	
1,1,1-Trichloroethane	
Trichloroethylene	
Methylene Chloride	
Miscellaneous:	
Benzene	
Ethyl acetate	
Ethanol	



**Figure 4.** VOCs detected in smoker's office versus nonsmoker's office (a) Reconstructed ion chromatogram from smoker's office. Collection on Tenax; analysis by TD/GC/MS. Peak identifications: A, carbon dioxide; B, 1,1,2-trichloro-1,2,2-trifluoro-ethane; C, pyrididine; D, hexane; E, ethyl acetate; F, 1,1,1-trichloroethane; G, benzene; H, trichloroethylene; I, toluene; J, trichloroethylene; K, 1,3,5-trimethylcyclohexane; L, o-xylene; M, nonane; N, dimethyloctane; O, propylenebenzene; (b) Reconstructed ion chromatogram from nonsmoker's office. Collection on Tenax, analysis by TD/GC/MS. Peak identifications: A, carbon dioxide; B, 3-methylpentane; C, hexane; D, ethyl acetate; E, 1,1,1-trichloroethane; F, cyclohexane; G, trichloroethylene; H, toluene; I, tetrachloroethylene; J, ethylbenzene; K, o-xylene; L, nonane; M, 2-ethoxyethoxyethanol; N, trimethyloctane; O, dimethyldecane; P, decahydronaphthalene.

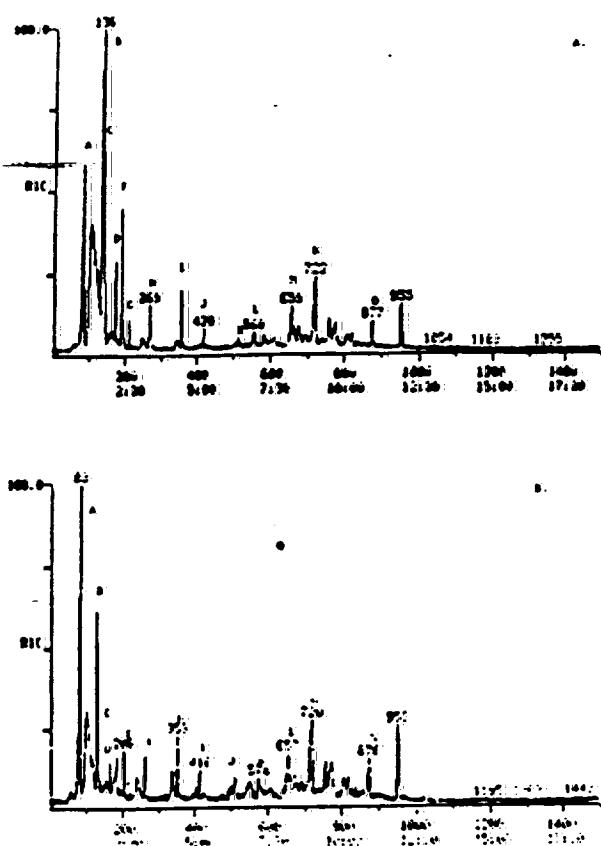
**Table 2.** Concentrations of selected VOCs detected in offices of smokers and nonsmokers

Compound	Concentration (ppbv)						
	Compound 1		Compound 2		Compound 3		
S	NS	S	NS	S	NS		
Toluene	20.9	28.6	68.6	30.1	0.16	0.24	
Ethyl acetate	35.3	62.6	24.2	104	nd	nd	
1,1,1-Trichloroethane	6.93	35.4	12.6	56.6	0.02	0.07	
Benzene	nd	2.53	8.86	nd	0.01	0.05	
Trichloroethylene	8.41	12.1	63.5	35.4	0.01	0.03	
Tetrachloroethylene	3.85	3.36	201	62.2	0.02	0.01	
Methylene chloride	63.4	nd	53.1	nd	nd	nd	
o-Xylene	2.85	nd	18.8	2.19	0.02	0.05	
Ethylbenzene	nd	nd	nd	5.28	0.01	nd	
Hexane	23.1	121	35.5	95.1	nd	nd	

S = Smoker

NS = Nonsmoker

2023380468



## REFERENCES

1. C. D. Hollowell and R. R. Miksch, *Bull N.Y. Acad. Med.* 57, 862 (1981).
2. National Research Council Committee on Indoor Pollutants, *Indoor Pollutants*, National Academy Press, Washington, D.C. (1981).
3. J. D. Spengler and K. Sexton, *Science* 221, 8 (1983).
4. L. Molhave, *Environmental International* 8, 117 (1982).
5. R. R. Miksch, C. D. Hollowell and H. E. Schmidt, *Environmental International* 8, 129 (1982).
6. L. Molhave, *Environmental International* 12, 167 (1986).
7. K. J. Krost, E. D. Pellezzi, S. G. Walburn, and S. A. Hubbard, *Analytical Chemistry* 54, 810 (1982).
8. R. G. Westendorf, *American Laboratory*, 72 (1985).
9. R. R. Arnts, *J. of Chromatog* 329, 389 (1985).
10. J. Sovcik, *American Laboratory*, 48 (1984).
11. G. Holzer, J. Oro and W. Bertsch, *J. of Chromatog* 126, 771 (1976).
12. R. F. Arrendale, R. F. Severson and O. T. Chortyk, *Beitr Tabakforsch* 12, 185 (1984).
13. C. Morris, R. Berkley, and J. Burgeiner, *Anal. Letters* 16, 1585 (1983).
14. V. Norman, *Recent Advances in Tobacco Smoke*. 31st Tobacco Chemists' Research Conference, Greensboro, North Carolina, 28 (1977).
15. R. F. Arrendale, W. J. Chamberlain, O. T. Chortyk, J. L. Baker and M. G. Stephenson, *Anal. Chem.* 58, 555 (1986).
16. K. El-Bayoumy, M. O'Donnell, S. S. Hecht and D. Hoffmann, *Carcinogenesis* 6, 805 (1985).
17. J. D. Adams, S. J. Lee and D. Hoffmann, *Carcinogenesis* 6, 221 (1985).
18. S. S. Hecht, C. B. Chen and D. Hoffmann, *Appl. of Chem. Research* 12, 82 (1979).
19. I. Schmetz, A. Wenger, D. Hoffmann and T. C. Too, *J. Agric. Food Chem.* 27, 802 (1979).
20. K. D. Brunnenmann, L. Yu and D. Hoffmann, *Cancer Res.* 37, 3218 (1977).
21. Y. Saint-Jalm and P. Moro-Toeta, *J. of Chromatog.* 190, 189 (1980).
22. I. Schmetz and D. Hoffmann, *Chemical Reviews* 77, 285 (1977).
23. J. L. Repace and A. H. Lowrey, *Science* 208, 484 (1980).
24. D. C. Wilkins, J. R. Whitaker and W. Jennings, *J. Chromatog. Sci.* 17, 269 (1984).
25. C. W. Bayer and M. S. Black, *Proceedings IAC'86, Managing Indoor Air for Health and Energy Conservation*, Atlanta, Georgia (1986), in press.
26. R. B. Griffith and R. Hancock, *Toxicology* 34, 123 (1985).

2023380470

# MEASUREMENTS OF ENVIRONMENTAL TOBACCO SMOKE IN AN AIR-CONDITIONED OFFICE BUILDING

C. J. Proctor\*, N. D. Warren and M. A. J. Bevan

BAT(UK & E) Research and Development Centre,  
Regents Park Road, Southampton, SO9 1PE, U.K.

(Received 12 May 1989; In final form 18 September 1989)

## ABSTRACT

This paper reports levels of nicotine, respirable particulates, carbon monoxide, carbon dioxide and volatile organic compounds measured in the air of smokers' and non-smokers' offices in a modern air-conditioned building. The results show very low levels of environmental tobacco smoke constituents, such as nicotine, present in smokers' offices. Moreover, the data show that smoking has little influence on the levels of volatile organic compounds found in the office air.

## INTRODUCTION

Environmental Tobacco Smoke, ETS, is the complex mixture of chemicals found in air as a specific result of smoking (1). Some reports have claimed that ETS is harmful to the health of the non-smoker (2,3,4). This issue has been discussed by scientists and doctors for over a decade, and although knowledge has increased over this period, it is still the subject of scientific controversy (2, 5). The claims have primarily been based on combining the results of epidemiological studies that have all been stated to be, when taken individually, inadequate (2, 3, 4, 6). Several experts in the field of low-risk epidemiology have stated that it is not possible to draw firm conclusions as to whether or not ETS is harmful to the health of the non-smoker (5, 6, 7).

In spite of the continuing debate, there have been calls for the introduction of further restrictions on where smoking can take place (4, 8). Much attention has recently focused on the work place, and in particular to the modern office environment.

Ever since the energy crisis of the 1970's, many of the office buildings constructed in the Western world have been designed with air conditioning systems that limit energy costs and assist in energy conservation. Often control over the amount of fresh air taken into the building is determined simply by the temperature measured at various points.

It has been well documented that buildings release chemicals into the air (9). Building materials, furnishings and coverings, and the building occupants will all contribute to the chemical burden of the office air (10). In extreme cases this may result in 'Sick Building Syndrome', where the occupants suffer symptoms and discomforts including headaches, burning eyes, irritation of the respiratory system, drowsiness, fatigue and general malaise (11). The causes of Sick Building Syndrome are

2023380471

not entirely defined, but are likely to be primarily exposure to bacteria, moulds and fungi produced and circulated by poorly maintained ventilation systems, and exposure to volatile organic compounds produced by various sources (12, 13).

The United States Environmental Protection Agency claimed, as part of the conclusions to their Total Exposure Assessment Methodology (TEAM) Studies, that the presence of ETS results in significantly higher levels of volatile organic compounds in air (14). Other authors, using similar methodologies, were unable to distinguish between smoking and non-smoking environments when measuring ambient volatile organics (15).

This paper presents the results from an investigation of the air in offices in a modern air-conditioned building located in Southern England. Ten offices, of different sizes, population and density of smokers, were each visited on five separate occasions. On each occasion, measurements were made of levels of nicotine, respirable suspended particulates, carbon monoxide, carbon dioxide and volatile organic compounds in the air.

### THE BUILDING

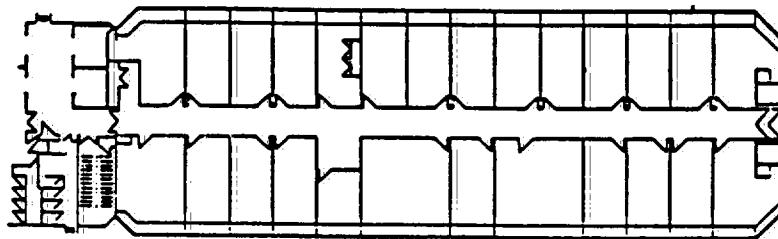
The building selected for this study is a 1970's built office block comprising of around 9300 square metres of floor space on 16 floors, and holding around 350 people. Although originally designed to be open plan, it has been modified over the years to incorporate a modular office design, though some open plan areas remain.

Air conditioning is nominally the same in all areas. There are two systems, one at the perimeter and one operating through the core of the building. Air for both systems is drawn from the roof where it is filtered and humidified. The perimeter system enters on each floor at vents positioned on window sills and exits through vents in the ceiling at the centre of the building. This system is monitored for temperature and relative humidity on three floors (floors 14, 11 and 5), conditions being fed-back to a central controlling system. The volumetric flow rate for the perimeter system was  $17.5 \text{ m}^3 \text{ s}^{-1}$ . With 544 vent outlets, the volumetric flow per module was  $1.93 \text{ m}^3 \text{ min}^{-1}$ , resulting in a typical air exchange rate for each office of around 3 air changes per hour.

The core system operates through the centre of each floor, often in a corridor, at a total volumetric flow rate of  $17 \text{ m}^3 \text{ s}^{-1}$  and is controlled by rheostats and motorised valves on every floor. A typical floor plan is given as Figure 1.

FIGURE 1

TYPICAL FLOOR PLAN OF THE BUILDING UNDER INVESTIGATION



Maximum possible recirculation is 84% (i.e. 16% incorporation of fresh air), though this condition is rarely used and recirculation rates vary throughout the day. The entire system is operated in a manner that minimises total energy costs.

### SAMPLING SITES

10 offices were selected to represent the variety of environments within the building. This included open plan space, single and multiple occupier offices, with different populations and numbers of smokers, as detailed in Table 1.

TABLE 1: Details of Sampling Sites

Site Number	Floor	Number of Occupants		Approximate Size (m <sup>3</sup> )
		Non-smokers	Smokers	
1	12	0	1	30
2	11	0	1	60
3	10	3	1	115
4	9	3	0	80
5	7	29	1	760
6	6	2	3	180
7	5	4	1	155
8	4	3	3	90
9	4	1	0	30
10	11	1	0	60

Samples were acquired between 0900 and 1600 hrs. Each site was visited 5 times, each occasion for a particular office being at a different time of the day and on different days to avoid any bias from possible temporal variations. So, for example, site number 7 was sampled between 14.40 and 15.40 on day 3, 10.20 and 11.20 on day 5, etc.

Each sample was acquired for one hour, and the sample was taken as near as possible to the centre of the office and at approximately head height of a seated person.

No smoker segregation is imposed in this building, and so smokers are free to visit and smoke in the offices of non-smokers. As a general rule, this rarely occurred in the sites investigated in this study.

### Analytical Considerations

All of the analytical methods for the analysis of the components under investigation have been previously detailed in the scientific literature. Briefly, the methods were:

- a) Nicotine: Airborne nicotine was collected by drawing, at a rate of 1 litre per minute, air through a sorbent sampling tube containing XAD-4 resin (20/40 mesh) (SKC, Inc.) (16, 17). After sampling, the tube was capped and returned to the laboratory. The collected nicotine was extracted from the resin using a quantity of ethyl acetate, modified with 0.01% triethylamine (to prevent losses of nicotine to the glassware). Analysis was effected by capillary gas chromatography with nitrogen-phosphorous detection. With this flow rate, and sampling periods of one hour, detection limits equate to approximately 0.1 µg m<sup>-3</sup> nicotine.

- b) Respirable suspended particulates (RSP): Airborne particulates were measured by gravimetric analysis. Air from the environment was drawn at 2 litres per minute through a fluoropore membrane filter (17) (Millipore UK Ltd) via an impactor separating at 3.5 microns. The filter was weighed on an electronic balance capable of resolving  $\pm 0.1 \mu\text{g}$  (Perkin-Elmer), before and after sampling, each time being conditioned first at 50% relative humidity, to arrive at the RSP measurement.
- c) Ultra-Violet Respirable Suspended Particulates (UV-RSP): In order to estimate the contribution of ETS to the total respirable particulates, each filter, after being weighed, was extracted with methanol and the resulting solution analysed for its absorbance at 325 nm. This was achieved by injecting the solution through a columnless liquid chromatography system into a UV detector and integrating the resultant peak. Previous research has shown that if only ETS is present (i.e. in controlled conditions) then the calculated UV-RSP value is equivalent to RSP when using 1,1,2,2-tetrahydroxybenzophenone as a surrogate for calibration (18). In real-world environments, UV-RSP will give an over-estimate of the ETS contribution, as other sources may contribute chemicals collected that also absorb at 325 nm (19).
- d) Carbon monoxide (CO): The CO monitoring system consists of a constant flow sampling pump and a detector based on electrochemical measurements (supplied by Neotronics, Gainesville, GA). Output from the detector was fed into a data logger (Campbell Scientific, Utah) which recorded signals every 30 seconds. Unlike all of the other measurements, CO analysis gave continuous real-time readings. Data given for each sample are the arithmetic mean of the readings over the sampling period.
- e) Carbon dioxide ( $\text{CO}_2$ ): Dräger tubes ( $\text{CO}_2$  0.01%/a, CH30 801) were used to measure ambient  $\text{CO}_2$  levels (Drägerwerk, West Germany). This measurement was made approximately 5 minutes prior to the end of each sampling period.
- f) Volatile organic compounds (VOC): Volatile chemicals present in the air were collected by drawing the air, using a fixed diaphragm pump at a rate of  $10 \text{ cm}^3$  per minute, through a stainless steel tube containing the absorbent Tenax TA (60 - 80 mesh) (20). After collection, each tube was capped and returned to the laboratory. Analysis of each sample required thermal desorption (using a Perkin-Elmer Ltd. ATD-50) for 20 minutes at  $240^\circ\text{C}$ , during which time the eluted chemicals were swept from the sampling tube to a cryofocusing trap maintained at  $-30^\circ\text{C}$  and containing a small quantity of Tenax. After this primary desorption the cold trap was rapidly heated electronically to  $240^\circ\text{C}$  whereby the chemicals were effectively injected in a narrow band onto a capillary gas chromatography column connected to an ion trap detector (a bench-top mass spectrometer supplied by Perkin-Elmer Ltd.). The capillary column (30m,  $0.32 \mu\text{m}$  ID, DB-5) separated the individual components, and the mass spectrometer both identified and quantified. For each chromatographic peak, compound identification was confirmed by its mass spectrum, and quantification used the base peak of the mass spectrum (e.g. benzene was quantified on the signal due to the m/z 78 ion). Calibration of this system required introduction of mixed standards of the compounds of interest injected at five different levels, each level in duplicate, onto a clean Tenax tube in order to run standards through the entire procedure. In order to check the performance of the instrument, a quantity of 2-bromonaphthalene (in methanol) was injected onto each tube prior to sampling. During sampling, the methanol eluted through the Tenax, but the 2-bromonaphthalene remained trapped for subsequent thermal desorption.

## RESULTS AND DISCUSSION

The data for all smokers' and non-smokers' offices are given in Table 2. Arithmetic means, medians, minimum and maximum values are given for each of the analytes. All data points from the total of 50 visits are included. Arithmetic means are generally of a higher value than medians due to a skewed distribution of values.

2023380474

TABLE 2

## SUMMARY OF DATA FROM ALL SAMPLING SITES

(Data in  $\mu\text{g m}^{-3}$  apart from CO and  $\text{CO}_2$  which is given in ppm)

	Site 1	Site 2	Site 3	Site 4	Site 5	Site 6	Site 7	Site 8	Site 9	Site 10
No. Smokers $\times 10^3 \text{ m}^{-3}$	33	17	8.7	0	1.3	17	6.5	33	0	0
NICOTINE	mean	8	18.1	3.6	0.2	1.2	6.9	2.1	2.5	0.5
	median	4.7	18.1	4.1	0.1	1.0	6.0	2.2	2.4	0.7
	range	2-19	10-26	1.5-5	0.1-0.5	0.6-2.1	4.2-11.4	1.2-3.1	0.7-4.7	0.1-0.8
RESPIRABLE SUSPENDED PARTICULATES	mean	97	148	91	116	97	109	80	101	52
	median	82	138	71	71	129	70	81	80	43
	range	41-167	91-225	40-172	69-208	19-150	43-210	49-118	67-199	27-91
UV-RSP	mean	23	61	7	11	8	30	13	18	3
	median	17	69	6	9	7	27	13	19	3
	range	1-75	29-72	1-17	2-15	2-14	21-48	9-19	4-28	1-6
CARBON MONOXIDE	mean	1.2	1.4	0.9	1.4	2.3	1.8	1.2	1.0	1.3
	median	1.0	1.4	1.0	1.0	1.6	1.2	0.9	1.0	1.0
	range	0.9-1.4	1.0-2.0	0.5-1.2	0.7-3.6	1.0-4.8	1.0-2.6	0.8-2.3	0.6-1.3	0.7-2.2
CARBON DIOXIDE	mean	730	570	520	610	560	600	610	540	520
	median	800	600	500	600	500	600	600	600	500
	range	450-1000	500-650	450-600	500-800	500-700	550-700	600-650	450-600	500-550
BENZENE	mean	10	8	7	12	21	19	15	9	15
	median	10	8	6	9	13	6	7	5	12
	range	5-14	5-13	3-14	3-23	6-49	6-48	5-46	5-21	9-31
CHLOROBENZENE	mean	0.2	0.2	0.2	0.3	0.6	0.4	0.4	0.3	0.7
	median	0.2	0.2	0.2	0.3	0.8	0.3	0.3	0.3	0.4
	range	0.1-0.4	0.1-0.4	0.1-0.5	0.1-0.4	0.1-1.0	0.1-1.0	0.1-1.1	0.1-0.6	0.3-2

1007

2023380475

TABLE 2 CONTINUED

1006

		Site 1	Site 2	Site 3	Site 4	Site 5	Site 6	Site 7	Site 8	Site 9	Site 10
N-DECANE	mean	6	8	8	5	9	12	7	6	10	4
	median	4	6	5	5	7	15	5	3	8	4
	range	4-13	1-18	1-24	1-7	2-22	2-20	2-16	3-13	6-16	1-6
1,2-DICHLORO-BENZENE	mean	0.3	0.4	0.4	0.7	0.7	0.5	0.4	0.4	0.6	0.7
	median	0.2	0.2	0.3	0.7	0.7	0.3	0.4	0.4	0.4	0.6
	range	0.2-0.6	0.1-0.9	0.2-0.8	0.2-1.1	0.2-1.4	0.1-1.1	0.1-0.7	0.2-0.5	0.2-1	0.4-1.0
1,2-DICHLORO-ETHANE	mean	11	9	13	12	14	19	16	8	17	15
	median	10	8	6	12	17	19	18	9	19	15
	range	6-16	6-13	4-37	5-18	5-24	5-39	4-21	3-12	10-27	5-19
DODECANE	mean	3	2	1	1	1	2	2	2	2	3
	median	2	2	1	1	1	2	1	1	2	1
	range	2-4	1-2	1-2	0.2-2	0.7-2	0.6-3	0.9-3	1-3	1-3	1-11
ETHYL-BENZENE	mean	5	4	3	5	11	54	6	4	5	5
	median	5	3	3	4	6	52	4	5	3	5
	range	3-6	3-8	2-4	1-13	3-26	11-122	3-15	3-5	3-13	4-7
LIMONENE	mean	7	4	3	2	5	10	7	3	4	2
	median	6	4	2	2	6	5	7	3	4	2
	range	4-11	2-8	2-8	0.4-4	1-9	4-31	5-8	2-6	2-8	1-3
N-OCTANE	mean	2	2	2	2	5	312	5	4	4	6
	median	2	2	2	2	3	485	2	5	4	2
	range	2-3	1-2	1-3	1-3	2-10	22-528	2-9	2-5	2-6	1-15
$\alpha$ -PINENE	mean	2	3	2	5	6	5	4	3	4	5
	median	2	2	2	4	6	3	4	3	3	4
	range	1-4	1-7	1-4	2-8	2-11	3-11	2-7	2-5	3-7	2-7
STYRENE	mean	6	9	7	11	27	17	19	10	26	16
	median	5	8	4	12	42	6	13	10	15	15
	range	2-16	4-17	3-21	6-16	4-50	4-53	3-59	3-20	7-79	4-28

2023380476

TABLE 2 CONTINUED

		Site 1	Site 2	Site 3	Site 4	Site 5	Site 6	Site 7	Site 8	Site 9	Site 10
TETRACHLOR-ETHENE	mean	2	3	2	3	3	3	3	3	4	3
	median	2	3	2	2	3	2	3	3	4	2
	range	1-3	1-6	1-4	1-8	1-5	1-5	2-6	1-3	3-5	1-5
TRICHLORO-ETHENE	mean	4	3	3	2	9	3	5	5	6	4
	median	4	3	2	1	5	2	1	4	4	1
	range	2-5	2-5	1-8	0.2-6	2-19	0.1-10	0.5-19	2-12	1-14	1-9
TOLUENE	mean	24	22	21	27	32	120	35	23	25	25
	median	24	22	23	20	25	113	22	22	22	20
	range	18-27	10-36	11-27	7-65	19-68	22-292	15-98	20-25	16-46	17-34
UNDECANE	mean	5	5	4	4	4	6	5	4	7	4
	median	4	6	4	4	3	7	4	3	7	4
	range	3-8	3-7	2-8	1-8	2-8	2-10	2-12	2-7	5-12	2-6
2-VINYL-PYRIDINE	mean	1	1	3	0.9	6	8	2	1	3	1
	median	0.5	0.3	2	0.3	2	0.6	2	0.9	1.4	0.2
	range	0.4-3	0.1-4	0.2-9	0.1-3.5	0.1-23	0.4-27	0.4-5	0.3-2	0.6-7	0.1-2
O-XYLENE	mean	7	8	6	11	24	33	12	9	11	14
	median	6	6	6	11	21	25	12	6	7	10
	range	4-11	4-15	3-9	5-22	8-50	10-68	4-24	5-19	5-27	6-25
M/P-XYLENE	mean	35	38	28	60	111	191	60	45	69	81
	median	26	24	17	66	109	149	60	33	37	89
	range	18-63	14-83	15-55	23-94	34-228	49-328	14-138	17-92	25-170	31-138
Pooled VOC's*	mean	139	148	117	164	291	822	206	141	214	201
	median	118	126	92	156	271	898	168	119	154	181

\*Pooled VOC's is the sum of the concentrations of all volatiles specifically identified in this study. It is not a measure of total volatiles.

202338047

In order to assess the worst and best air quality for both smoking and non-smoking situations, Table 3 presents individual values for the minimum and maximum RSP observed for smokers' offices and for non-smokers' offices. So, for example, the 'minimum smokers' offices' data in the first column presents all of the analyte values for the single visit that gave the minimum RSP value ( $40 \mu\text{g m}^{-3}$ ). For this, data from Site 5, which is an open-plan space with one smoker and twenty-nine non-smokers, has been excluded from the considerations in order not to bias the comparisons.

Table 4 presents the same evaluation, except that it is based on the single visit corresponding to the minimum and maximum CO value observed.

The data is best evaluated by considering each analyte, or group of analytes, in turn.

### Nicotine

In smokers' offices the median airborne nicotine level was  $3.1 \mu\text{g m}^{-3}$  (range  $0.6$  to  $26 \mu\text{g m}^{-3}$ ). From Table 2 it can be seen that the highest median for an individual site was  $18.1 \mu\text{g m}^{-3}$  for Site 2, whilst the lowest median was  $1.0 \mu\text{g m}^{-3}$  for Site 5. The nicotine data is of a similar order, though slightly lower in magnitude, as compared to other studies of offices (19, 21, 22). Numbers of cigarettes smoked during each visit were not identified in this study, because we did not wish to influence the occupants behaviour by either observing or questioning. The data show little correlation between nicotine levels and numbers of smokers present, or between nicotine levels and smoker density (the number of smokers present divided by the size of the room).

Some nicotine was found in the air of non-smokers' offices, though this was at a low level with a median value of  $0.6 \mu\text{g m}^{-3}$  (range  $0.1$  to  $2.1 \mu\text{g m}^{-3}$ ). Site 4 gave a median nicotine value of  $0.1 \mu\text{g m}^{-3}$ , whilst Site 10 gave a value of  $1.4 \mu\text{g m}^{-3}$ . Site 10 had been occupied by a smoker up to one week prior to the start of this investigation and hence it is possible that this level is due to a re-emission of nicotine from the furnishings. It is also possible that some nicotine is transferred through the air-conditioning system, or that unknown to us, a smoker occasionally visited this site.

### RSP and UV-RSP

The RSP median value in smokers' offices was  $91 \mu\text{g m}^{-3}$  (range  $19$  to  $225 \mu\text{g m}^{-3}$ ). The UV-RSP median value, which is an estimate of the possible ETS contribution to RSP, was  $24 \mu\text{g m}^{-3}$  (range  $1$  to  $75 \mu\text{g m}^{-3}$ ).

Median values of RSP for each smoking site correlated poorly (0.522) with corresponding nicotine values. However, median UV-RSP values, again for each smoking site, gave an excellent correlation (0.962) with corresponding nicotine values. This indicates that the sum of the other particulate sources in this building is far more significant than the contribution from ETS (which may be, in smokers' offices, of the order of 30% of the total).

Data from non-smokers' offices yields a median RSP value of  $71 \mu\text{g m}^{-3}$  (range  $27$  to  $208 \mu\text{g m}^{-3}$ ). Some UV-RSP was also observed, with a median of  $8.8 \mu\text{g m}^{-3}$  (range  $1$  to  $17 \mu\text{g m}^{-3}$ ). The non-smoking Site 10 had a higher median RSP than smoking Sites 1, 3, 4, 6, 7 and 8, though the median UV-RSP for Site 10 was only higher than smoking sites 3 and 5.

Interestingly, Site 5 (the open plan area with 29 non-smokers and one smoker) had a higher than average median RSP value ( $129 \mu\text{g m}^{-3}$ ) but a lower than average UV-RSP value ( $7 \mu\text{g m}^{-3}$ ), indicating a significant non-ETS particulate source in this area.

The comparison of minimum and maximum RSP visits for smokers' and non-smokers' offices (Table 3) is interesting. Overall, there is a tendency for analyte levels to increase corresponding to the increase in RSP value. The increase is not of the same order of magnitude (apart from nicotine and UV-RSP in smokers' offices). Comparing benzene levels, for example, there is an increase from  $4$  to  $8 \mu\text{g m}^{-3}$  in smokers' offices, and an increase from  $15$  to  $18 \mu\text{g m}^{-3}$  in non-smokers' offices.

**TABLE 3:** Data from a single visit corresponding to the minimum and the maximum RSP value obtained\*, separate by smokers' and non-smokers' offices.  
 (Data in  $\mu\text{g m}^{-3}$ , apart from CO and  $\text{CO}_2$ , which is given in ppm)

	Smokers' Offices		Non-Smokers' Offices	
	Minimum (Site 3)	Maximum (Site 2)	Minimum (Site 9)	Maximum (Site 4)
Nicotine	1.5	11	0.3	0.1
Respirable suspended particulates (RSP)	40	225	27	208
UV-RSP	3	72	3	4
Carbon monoxide	0.8	1.0	2.2	1.0
Carbon dioxide	550	500	500	600
Benzene	4	8	15	18
Chlorobenzene	0.1	0.3	0.4	0.3
n-Decane	24	4	8	5
1,2-Dichlorobenzene	0.2	0.5	0.4	0.7
1,2-Dichloroethane	4	10	20	18
Dodecane	2	2	1	2
Ethyl benzene	3	8	3	5
Limonene	3	4	4	4
n-Octane	3	2	4	2
$\alpha$ -Pinene	1	7	3	8
Styrene	4	15	10	12
Tetrachloroethylene	3	6	4	8
Trichloroethylene	2	5	1	0.2
Toluene	11	36	23	25
Undecane	8	3	6	4
2-Vinyl Pyridine	0.5	0.3	1.3	0.3
o-Xylene	6	15	7	11
m/p-Xylene	16	83	33	81

\* Site 5 has been excluded from consideration

2023380479

**TABLE 4:** Data from a single visit corresponding to the minimum and maximum CO value obtained\*, separated by smokers' and non-smokers' offices.  
 (Data in  $\mu\text{g m}^{-3}$ , apart from CO and  $\text{CO}_2$  which is given in ppm)

	Smokers' Offices		Non-Smokers' Offices	
	Minimum (Site 3)	Maximum (Site 6)	Minimum (Site 9)	Maximum (Site 4)
Nicotine	2.0	4.2	0.8	0.1
Respirable suspended particulates (RSP)	50	33	43	71
UV-RSP	6	21	6	9
Carbon monoxide	0.5	3.4	0.7	3.6
Carbon dioxide	600	700	500	800
Benzene	6	48	12	23
Chlorobenzene	0.2	0.5	0.6	0.4
n-Decane	2	20	7	5
1,2-Dichlorobenzene	0.2	0.1	0.9	1.1
1,2-Dichloroethane	4	39	19	12
Dodecane	1	3	3	1
Ethyl benzene	2	54	4	13
Limonene	2	4	5	2
n-Octane	2	485	5	2
$\alpha$ -Pinene	1	6	6	7
Styrene	3	15	19	16
Tetrachloroethylene	1	5	4	4
Trichloroethylene	0.5	10	4	1
Toluene	22	292	22	65
Undecane	2	10	7	8
2-Vinyl Pyridine	9	0.6	3	4
$\text{o}$ -Xylene	4	41	11	22
$\text{m/p-Xylene}$	17	149	68	94

Repace and Lowrey (23) have suggested that a typical non-smoker working in an office building in the U.S. (generally air-conditioned), would be exposed to average concentrations of particulates due specifically to ETS of  $242 \mu\text{g m}^{-3}$  (range 100 to  $1000 \mu\text{g m}^{-3}$ ). On this basis, these authors proposed numbers of deaths in non-smokers due to exposure to ETS in the workplace and suggested that workplace smoking restrictions should be introduced. Our data, acquired in a relatively well ventilated but not untypical U.K. office building, suggests average ETS particulate levels some 10 times lower than those given by Repace and Lowrey; UV-RSP values in our study for smokers' offices being  $24 \mu\text{g m}^{-3}$  (range 1 to  $75 \mu\text{g m}^{-3}$ ).

It is known that Repace and Lowrey did not take into proper account particulate sources other than ETS, but rather suggested that ETS was the major source of particulates. Our study suggests that this might not be so. The work of other researchers in the U.S. also suggests that the Repace and Lowrey data may be a gross over-estimate (24).

#### Carbon Monoxide

Median carbon monoxide levels were 1.1 ppm (range 0.5 to 3.4 ppm) for smokers' offices, and 1.0 ppm (range 0.7 to 3.6 ppm) for non-smokers' offices.

Comparing median values for each office, Site 5 has the highest at 1.6 ppm. This is associated with a relatively high RSP value and a relatively low UV-RSP value for this Site, indicating a source other than ETS being responsible. All other sites were found to have similar median CO levels (range 0.9 to 1.4 ppm).

For smokers' offices alone, and excluding Site 5, there was a relatively good correlation between median CO level for each site and both the corresponding nicotine level (corr. 0.929) and the corresponding UV-RSP level (corr. 0.924). This correlation was not so well defined for corresponding RSP values (corr. 0.752). This suggests that ETS is contributing to the CO level in smokers' offices, though this contribution seems to be of the order of 0.1 to 0.4 ppm.

Table 4 compares four individual visits corresponding to minimum and maximum CO values for smokers' and non-smokers' offices. For smokers' offices the increase in CO from minimum to maximum corresponds to increases in many of the other analytes. However, Site 6 (where the maximum CO level was observed) was a drawing office where many print materials were being used, and so this confounds the interpretation. For non-smokers' offices, the CO level increase from minimum to maximum corresponded to an increase in particulate,  $\text{CO}_2$  and some (but not all) aromatic VOC levels, but a decrease in hydrocarbons and in some chlorinated VOCs.

The relatively low CO levels observed may, in part, be due to the fact that air is drawn into the building from roof level, well away from the traffic circulating the building.

#### Carbon Dioxide

Median  $\text{CO}_2$  levels for all smokers' sites was 600 ppm (range 450 to 1000 ppm) and 500 ppm (range 450 to 800 ppm) for non-smokers' offices. These levels suggest that the building is relatively well ventilated.

Comparing medians for each site office, Site 1 (a small office occupied by a single smoker) consistently had higher levels of  $\text{CO}_2$  than other sites. A comparison of Site 1 with Site 9 (a similarly small office occupied by a single non-smoker) suggest that the high  $\text{CO}_2$  levels in Site 1 are not a simple correlation with size of room. It was noticed, however, that in Site 1, ventilation inlets at the window sills were significantly obstructed and this may be of importance.

## Volatile Organic Compounds

Some 18 VOC's were quantified in this study. One of the most interesting is benzene. The EPA's TEAM study (14) has suggested that ETS is a major source of benzene in indoor air. Our data from this office building does not confirm that suggestion. The median benzene level in smokers' offices was  $8 \mu\text{g m}^{-3}$  (range 3 to  $49 \mu\text{g m}^{-3}$ ), whilst the median benzene level in non-smokers' offices was  $10 \mu\text{g m}^{-3}$  (range 3 to  $31 \mu\text{g m}^{-3}$ ). The difference in benzene levels between smokers' and non-smokers' offices was not statistically significantly different at the 95% confidence limit.

It is found that there is poor correlation over individual smokers' offices (excluding Site 5) between median benzene levels and median nicotine (corr. 0.324) or between median benzene levels and median UV-RSP (corr. 0.243). Looking across all offices, Site 5 has the highest median benzene level ( $13 \mu\text{g m}^{-3}$ ) with Site 9 the second highest ( $12 \mu\text{g m}^{-3}$ ).

The absolute levels of benzene found in this study are similar to that found in the TEAM study, but the conclusions on what is the major source of benzene are quite different.

Comparing all of the median VOC values in Table 2, 9 VOC's are higher in non-smokers' offices, 7 are higher in smokers' offices and 2 are similar in both situations. However, levels are very similar for all VOC's comparing smoking and non-smoking offices. There is no indication that the presence of ETS is associated with significantly increased levels of VOC's in this office environment.

Comparing median values across individual sites, two offices stand out as being unusual. Site 5, which has relatively high RSP and CO values and low nicotine and UV-RSP values, has higher than average levels of benzene, styrene, o-and m/p-xylene, and chlorobenzene. No obvious source for these levels was identified (tobacco smoking was clearly not a major source). It is possible that this open plan area was not ventilated as efficiently as the smaller offices, though  $\text{CO}_2$  levels do not confirm this.

Site 6 is also unusual. Much higher than average levels of n-decane, n-octane, ethylbenzene, toluene and xylenes were identified. The source of these chemicals is clear. This site is a drawing office working with various inks and printing material, and located adjacent to rooms processing photographic materials. Hence when pooled VOC's are calculated by adding the median concentrations of all the VOC's (including nicotine) together, Site 6 is seen to contain around 9 times more VOC's than many of the other offices.

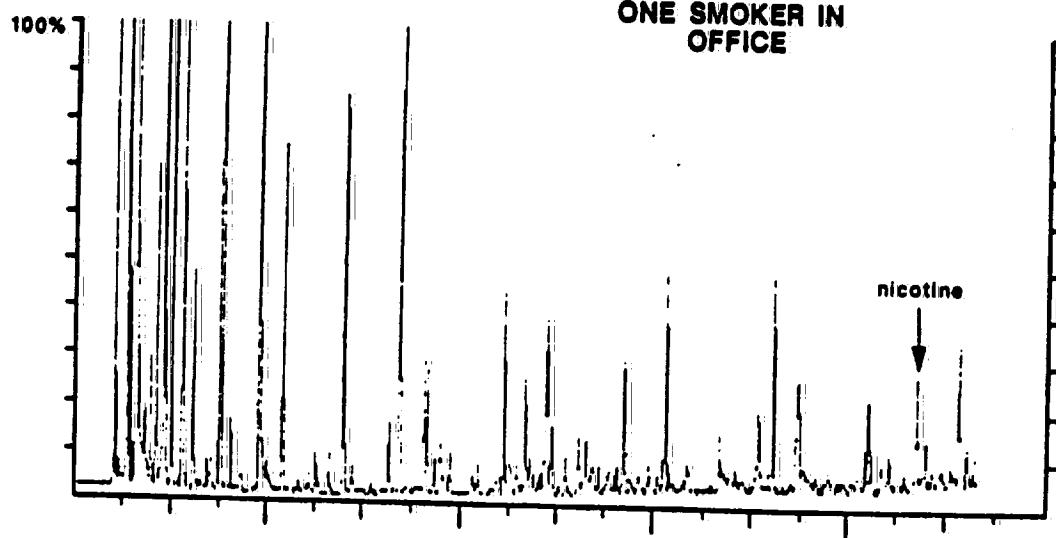
It should also be noted that the peak toluene value averaged over one of the hour long visits to Site 6 was a quarter of the odour detection limit, and the peak styrene level of  $79 \mu\text{g m}^{-3}$  was equivalent to the odour detection limit. This styrene level was observed on just one occasion in Site 9 (non-smoking; single occupant) and was also associated with higher than averaged levels of benzene, chlorobenzene, ethylbenzene, toluene and xylenes.

In order to illustrate the number of volatile chemicals present in the air of smokers' and non-smokers' offices, Figure 2 compares chromatographic profiles taken in Site 3 and Site 4. The two chromatograms may be directly compared as the 100% ion count has been adjusted for the volume of air sampled in each visit. It is clear that the chromatograms are similar, apart from the peak corresponding to nicotine (representing  $5 \mu\text{g m}^{-3}$ ) being present in the sample from the smokers' office.

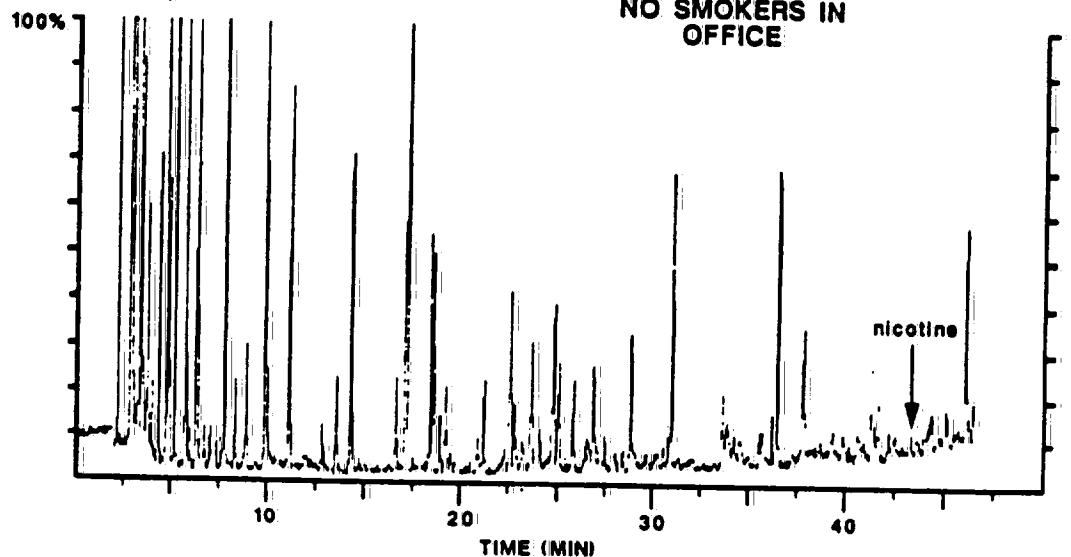
Figure 2:

CHROMATOGRAPHIC PROFILES OF  
MULTIPLE OCCUPANT OFFICES

TOTAL ION COUNT



TOTAL ION COUNT



1015

2023380483

### Cigarette Equivalent Calculations

Some authors (17, 19), have attempted to put the levels of ETS constituents into perspective through cigarette equivalent calculations. This exercise takes the median value of a constituent such as nicotine or UV-RSP and assumes that this is the constant exposure. A typical breathing rate and a time of exposure (e.g. the time spent in the office each day) is then used to arrive at a daily exposure to the constituent. This is then compared to the delivery of the relevant constituent (nicotine or particulate matter) that would be obtained from smoking one cigarette. Such calculations are strictly an estimate of exposure and not dose, are only relevant to the quantified constituent and not total ETS, and take no account of the differences between breathing air and inhaling smoke. However, with these facts noted, the calculations are still of interest.

So, if we assume a typical breathing rate at light work for a male adult of  $1.08 \text{ m}^3 \text{ h}^{-1}$  and for a female adult of  $0.62 \text{ m}^3 \text{ h}^{-1}$  (24, 25), an exposure time of 7 hours per day, 5 days per week, and typical mainstream deliveries from U.K. style cigarette of 1.3 mg per cigarette nicotine and 13.6 mg per cigarette particulates, then cigarette equivalent calculations can be made.

For nicotine, taking the median airborne nicotine value for smokers' offices as  $3.1 \mu\text{g m}^{-3}$ , then a non-smoker present all day in the office would, on this average, be exposed to the equivalent of 0.018 of a cigarette (male) or 0.010 of a cigarette (female). This means that a male non-smoker would have to work in the smoker's office for over 11 weeks before being exposed to the equivalent nicotine as from smoking one cigarette. For females, this time would have to be 20 weeks. In other words, a female non-smoker would have to work for over seven and one half years in the smoker's office before being exposed to the equivalent nicotine of smoking a pack of 20 cigarettes.

This is based on the median value. Even for the office with the highest median airborne nicotine (Site 2,  $18.1 \mu\text{g m}^{-3}$  nicotine), the nicotine cigarette equivalent values are 0.105 cigarette per day (male) or 0.06 cigarette per day (female).

If the calculation is based on UV-RSP as being an estimate of the ETS contribution to respirable particulates and taking the median value from smokers' offices as  $24 \mu\text{g m}^{-3}$ , then the non-smoker working all day in the smoker's office would be exposed to the equivalent particulates of 0.013 (male) or 0.0077 (female) cigarettes per day. This again would result in a male non-smoker working in the smokers' office for 15 weeks before being exposed to the equivalent particulates as smoking one cigarette. For females this equates to almost 26 weeks.

The highest median UV-RSP (Site 2,  $69 \mu\text{g m}^{-3}$ ) results in particulate cigarette equivalents of 0.038 cigarettes per day (male) or 0.022 cigarettes per day (female).

These calculations simply serve to illustrate that levels of ETS constituents in the smokers' offices of this relatively well ventilated building are extremely small.

### CONCLUSIONS

This investigation of chemicals in the air of smokers' and non-smokers' offices in an air-conditioned building results in several conclusions.

1. The levels of constituents related to ETS (nicotine and UV-RSP) in smokers' offices were found to be low, both in terms of industrial time weighted exposure limits and in comparison to other chemicals present in the air.
2. In cigarette equivalent terms, a non-smoking male would have to work in a smoker's office for an average 11 weeks before being exposed to the nicotine equivalent of one cigarette. Based on particulates, this time extends to 15 weeks. For females, this equates to 20 weeks (based on nicotine) or 26 weeks (based on particulates).

3. Both medians and ranges of ETS-related particulate levels are some ten times lower than those suggested by Repace and Lowrey (23) to be typical of U.S. office buildings.
4. By comparing smokers' and non-smokers' offices, and by observing values obtained for RSP and UV-RSP, it seems that, in this environment, ETS was a minor contributor to respirable particulate levels in air.
5. The presence of ETS resulted in a slight increase in CO levels, of the order of 0.1 ppm.
6. ETS did not significantly contribute to levels of volatile organic compounds in office air. In direct conflict with the findings of the U.S. EPA TEAM study (14), our research suggests that tobacco smoking does not result in significant increases of compounds such as benzene in office air.

## REFERENCES

1. C.W. Nystrom and C.R. Green, Proceedings of the ASHRAE Conference IAQ86, Managing Indoor Air for Health and Energy Conservation, Atlanta, Georgia, April 20-23, 1986, 213-233.
2. Committee on Passive Smoking, Board of Environmental Studies and Toxicology, National Research Council, Environmental Tobacco Smoke. Measuring Exposures and Assessing Health Risks, National Academy Press, Washington DC, 1986.
3. Report of the U.S. Surgeon General, Health Consequences of Involuntary Smoking, U.S. Government Printing Office, Washington D.C., 1986.
4. The Fourth Report of the Independent Scientific Committee on Smoking and Health, Her Majesty's Stationery Office, London, 1988.
5. American Cancer Society, General Facts on Smoking and Health, 3, 1988.
6. K. Uberla, Int. Arch. Occup. Environ. Health, 9, 21-37, 1987.
7. P.N. Lee, Misclassification of Smoking Habits and Passive Smoking. A Review of the Evidence, Springer-Verlag, Berlin, 1988.
8. T.D. Sterling and B. Mueller, Am. Ind. Hyg. Assoc. J., 49, 423-426, 1988.
9. K. Sexton and J.J. Wesolowski, Environ. Sci. Technol., 19, 305-309, 1985.
10. National Research Council, Board of Toxicology and Environmental Health Hazards, Indoor Pollutants, National Academy Press, Washington D.C., 1981.
11. A.S. Robertson, P.S. Burge, A. Hedge, J. Sims, F.S. Gill, M. Finnegan, C.A.C. Pickering and G. Dalton, B.M.J. 291, 373-376, 1985.
12. E. Noma, B. Berglund, U. Berglund, I. Johansson, and J.C. Baird, Atmos. Environ., 22, 451-460, 1988.
13. M.J. Finnegan, C.A.C. Pickering and P.S. Burge, B.M.J. 289, 1573-1575, 1984.
14. L.A. Wallace, E.D. Pellizzari, T.D. Hartwell, C. Sparacino, R. Whitmore, L. Sheldon, H. Zelon and R. Perritt, Environ. Res., 43, 290-307, 1987.
15. C.W. Bayer and M.S. Black, J. Chrom. Sci., 25, 60-64, 1987.
16. M.W. Ogden, L.W. Eudy, D.L. Heavner, F.W. Conrad, and C.R. Green, presented at 40th Tobacco Chemists' Research Conference, Knoxville, TN, Oct 1986.

2023380485

17. G.B. Oldaker III, and F.C. Conrad, Environ. Sci. Technol., 21, 994-999, 1987.
18. M.W. Ogden, 101st Annual International Meeting of The Association of Official Analytical Chemists, Sept 14-17, San Francisco, CA, Poster H240, 1987.
19. J.R. Carson and C.A. Erikson, Environ. Tech. Lett., 9, 501-508, 1988.
20. C.J. Proctor, Environ. Tech. Lett., 9, 553-562, 1988.
21. T.D. Sterling, H. Dimich and D. Kobayashi, J. Air Pollut. Control Assoc., 32, 250-259, 1982.
22. S.K. Hammond, B.P. Leaderer, A.C. Roche and M. Schenker, Atmos. Environ., 21, 457-462, 1987.
23. J.L. Repace and A.H. Lowrey, Environ. Int., 11, 3-22, 1985.
24. A. Arundell, T. Sterling and J. Weinkam, Indoor and Ambient Air Quality, ed. R. Perry and P.W. Kirk, Seiber Press, 1988, 242-251.
25. P.L. Altman and D.S. Ditmer, Respiration and Circulation, Federation of American Society for Experimental Biology, Bethesda, MD, 1971.

2023380486

## PUBLIC HEALTH BRIEFS

cause of the incident, container structure and failure, and deaths and injuries resulting from the cargo. DOT attempts to validate death and injury data. Specifically excluded from reporting requirements are releases of small quantities of certain consumer commodities, and releases from motor carrier firms doing solely intrastate business and from certain water transporters. Automobiles striking storage tanks and certain transportation-related spills at fixed facilities are also excluded.

The Acute Hazardous Events (AHE) data base was begun in 1983 and uses the NRC as its main source of data. However, data are also included from selected state governments, the Environmental Protection Agency (EPA) Region 7, some newspapers and wire services, and other sources. Information collected includes cause of event, activity taking place during the event, and type of property damaged. Attempts are made to eliminate deaths and injuries not caused by hazardous materials. Because emphasis was placed upon events involving chemicals covered by Superfund legislation and air releases from fixed sites, many events which appear in the NRC data base are excluded. AHE is maintained and augmented by EPA and its contractors, primarily Industrial Economics, Incorporated, and has been updated through 1986.\* If events

appear in more than one source, they are checked for consistency; otherwise, data are not validated.

## REFERENCES

1. SAS Institute Inc: SAS User's Guide: Basics, Version 5 Ed. Cary, NC: SAS Institute Inc, 1983.
2. US Congress, Office of Technology Assessment: Transportation of hazardous materials. Washington, DC: Govt Printing Office, 1986: 65-86.
3. Shaw GN, Windham GC, Leonard A, Neura RN: Characteristics of hazardous material spills from reporting systems in California. *Am J Public Health* 1986; 76:540-543.
4. Industrial Economics, Inc: Management Technology and Data Systems, Inc; PEI Associates, Inc; ICF, Inc: Acute hazardous events data base. Springfield, VA: National Technical Information Service, 1985; EPA Contract No. 68-02-4055.

## Formaldehyde Exposures from Tobacco Smoke: A Review

THAD GODISH, PhD

**Abstract:** Reports of formaldehyde levels in mainstream, sidestream, and environmental tobacco smoke from nine studies are reviewed. Considerable disparity exists between formaldehyde production rates determined from mainstream-sidestream studies and those reporting levels in environmental tobacco smoke. Tobacco smoke does not appear to increase vapor-phase formaldehyde levels significantly in indoor environments, but formaldehyde exposure increases in the presence of cigarette smoke. (See *American Journal of Public Health* 1989; 79: 1044-1045.)

### Introduction

Formaldehyde is a major oxidation by-product of combustion processes including tobacco smoking. It is produced in both the mainstream (MS), and sidestream smoke (SS), and has been reported in substantial levels in environmental tobacco smoke (ETS).

Formaldehyde levels in mainstream, sidestream, and environmental tobacco smoke reported by a number of investigators are summarized in Table 1. Reported studies vary in testing methodologies and expression of concentrations. Concentration units are those originally reported and those calculated and standardized by the author from original data, assuming a smoking rate of 35 ml/puff and 10 puffs/cigarette.

As seen in Table 1, formaldehyde concentrations in mainstream smoke<sup>1-4</sup> ranged from about 10 µg/cigarette to over 100 µg/cigarette. Differences in concentrations reflect differences in tobacco type and brand. Higher average concentrations reported by the Surgeon General in 1986<sup>5</sup> reflect those of regular non-filter cigarettes.

Sidestream vapor-phase formaldehyde concentrations also varied somewhat. Ayer and Yeager<sup>6</sup> reported 15-48 ppm. Hoffman's observations ranged from nondetectable to 34.2 µg/

cigarette, with an average of 12.1 µg/cigarette for 16 different brands.<sup>4</sup>

Room or large chamber formaldehyde levels associated with environmental tobacco smoke<sup>6-9</sup> indicate that formaldehyde concentrations in such rooms are high. For example, in the studies of Howlett, *et al.*<sup>8</sup> one cigarette smoked in an environmental chamber caused the formaldehyde level to increase to 0.21 ppm within a half hour. Formaldehyde production rates calculated from ETS concentrations (Table 1) are substantially higher (one to two orders of magnitude) than those reported for MS, SS, and MS-SS combined.

The considerable disparity in formaldehyde production rates determined from MS-SS and ETS studies suggests differences due to methodologies employed in sampling and analysis. In the mainstream-sidestream smoke studies reported by the Surgeon General<sup>2,3</sup> and by Hoffman,<sup>4</sup> gas and particulate phase materials were separated by high-efficiency filtration. In studies by Weber, *et al.*<sup>6</sup> no attempt was made to remove particulate phase materials. Sundin<sup>7</sup> employed particulate phase filtration of unknown efficiency. Attempts to remove particulate phase materials in ETS samples were not reported by Howlett, *et al.*<sup>8</sup> and Klus, *et al.*<sup>9</sup>

In mainstream-sidestream smoke studies,<sup>2-4</sup> smoke samples were analyzed by the 2,4 dinitrophenylhydrazene-HPLC method which is specific for free formaldehyde. The chromotropic acid method<sup>10</sup> on the other hand was used in the studies of Weber, *et al.*<sup>6</sup> and Sundin<sup>7</sup>; it is likely to have been employed in the two other environmental tobacco smoke studies as well because it is the dominant method used to determine formaldehyde concentrations in air. In the chromotropic acid method, formaldehyde forms a stable addition product on sample collection in sodium bisulfite solution. On analysis, the addition product is destroyed yielding free formaldehyde. Any solution which contains free formaldehyde, a formaldehyde addition product, or organic compounds which produce formaldehyde on sulfuric acid destruction will test positive for formaldehyde.

On analysis with the chromotropic acid method, the particulate phase of tobacco smoke has been shown to contain appreciable quantities of formaldehyde.<sup>11</sup> This formaldehyde may be present as free formaldehyde dissolved in liquid water or it may be produced by the destruction of formaldehyde addition products and possibly other organic

\*Address reprint requests to Thad Godish, PhD, Indoor Air Quality Research Laboratory, Ball State University, Muncie, IN 47306. This paper, submitted to the Journal September 4, 1988, was revised and accepted for publication December 29, 1988.

© 1989 American Journal of Public Health 0090-0036/89/01044-02\$00.50/0

2023380182

TABLE 1—Formaldehyde Concentrations in Mainstream, Sidestream, and Environmental Tobacco Smoke

Mainstream		Sidestream		Environmental Tobacco Smoke		Cited Reference
μg/puff	ppm/puff	μg/cig.	ppm/cig.	ppm vol.	μg/cig.*	
1.7	40	17				1
1-8	23-210	10-90				2
7-10	188-234	70-100				3
0.9-11.9	22-273	8.2-118.9	N.D.-34.9			4
(7.4)	(177)	(74.2)	(12.1)			
			15-48			5
				1629		6
				731		7
				740		8
				641		9

\*Calculated from data derived from large chamber studies.

compounds in the solid or liquid phase. Because it is incorporated into their molecular structure, addition products are unlikely to yield significant quantities of free formaldehyde under normal circumstances. Formaldehyde dissolved in water can vaporize from the particulate phase, can remain in solution and react with other particulate phase compounds, or can remain in solution as free formaldehyde and then react with body tissues on inhalation. The potential for particulate phase tobacco smoke to produce and release free formaldehyde is unknown, and the health consequences of particulate phase materials are also unknown.

**Effect of Tobacco Smoking on Indoor Formaldehyde Levels**—Because of uncertainties associated with formaldehyde in the particulate phase of tobacco smoke samples and the lack of specificity of the chromatropic acid method for free formaldehyde, reported ETS studies are of limited usefulness in assessing the effect of tobacco smoking on vapor-phase formaldehyde levels in indoor air. Formaldehyde concentrations in indoor air can, however, be calculated from production rates reported by the Surgeon General<sup>1,2</sup> and by Hoffman.<sup>4</sup> The following "worst case" conditions are assumed: 1) 20 cigarettes/30 minutes smoking rate; 2) production rates of 120 and 34 μg/cigarette for MS and SS; 3) MS formaldehyde not retained by smoker; 4) zero air exchange rate in a 30 m<sup>3</sup> environmental chamber; and 5) no sinks or other sources present. Under these assumptions, 3080 μg formaldehyde would be produced resulting in a concentration of 0.085 ppm. In a 194 m<sup>3</sup> space (typical of a single-wide mobile home) the concentration would be considerably lower (0.012 ppm). Even under these extreme circumstances, the effect of cigarette smoking on formaldehyde levels in indoor spaces would be negligible. This is consistent with the residential measurements of Dally, *et al.*,<sup>11</sup> and Ritchie and Lehnen.<sup>12</sup>

**Exposures to Smokers**—Formaldehyde levels in mainstream smoke appear to be high. When dilution inspiration is taken into consideration, formaldehyde exposures on a per puff basis appear to be in the range of 1.5-19.5 ppm/puff. The cumulative daily exposure duration for a single pack/day consumption would be approximately 6 minutes and 40 seconds; the cumulative daily dose, 188-2382 μg (depending on brand smoked). Exposures at the upper end of the range for a one pack/day consumption would be approximately equivalent to an exposure of 0.25 ppm 22 hours/day in a mobile home environment.

**Health Risks**—Recent epidemiological studies indicate that formaldehyde exposures associated with residential environments are great enough to cause a variety of acute symptoms.<sup>13-15</sup> Formaldehyde may also cause asthma.<sup>16,17</sup>

In addition, the US Environmental Protection Agency (USEPA) has concluded that sufficient evidence exists to implicate formaldehyde as a human carcinogen.<sup>18</sup> USEPA risk assessments predict that individuals with average exposures of 0.16-0.19 ppm 16 hours/day for 10+ years in a mobile home have upperbound risks of  $1.5-3.40 \times 10^{-4}$ . Mainstream tobacco smoke exposures would be expected to confer their own formaldehyde cancer risk and to increase the risk of upper respiratory system cancer associated with exposures to formaldehyde-contaminated indoor air.

#### REFERENCES

- Kensler CI, Battista SP: Components of cigarette smoke with cilary-depressant activity. *N Engl J Med* 1963; 269:1161-1166.
- USDHEW: Smoking and Health: A Report of the Surgeon General. Pub. No. (PHS) 78-50069. Washington, DC: Govt Printing Office, 1979.
- USDHHS: The Health Consequences of Involuntary Smoking: A Report of the Surgeon General. Pub. No. (CDC) 87-8398. Washington, DC: Govt Printing Office, 1986.
- Hoffman DJ: Formaldehyde in cigarette main- and sidestream smoke. Valhalla, NY: Naylor Dame Institute for Disease Prevention, American Health Foundation, 1983.
- Ayer HD, Yeager DW: Irritants in cigarette smoke plumes. *Am J Public Health* 1962; 52:1123-1125.
- Weber A, Jermiin C, Grandjean E: Irritating effects on man of air pollution due to cigarette smoke. *Am J Public Health* 1976; 66:672-676.
- Sundin B, Hamro P: Formaldehyde emission from particle board and other building materials: A study from the Scandinavian countries. Makosy T (ed): Proc Twelfth Annual Particle Board Symposium—WSU, 1978; 251-273.
- Howlett CT, Newton LR, Stevens KR: Formaldehyde: A case study. Proc Fourth Int Conf Indoor Air Quality and Climate 1987; 2:676-681.
- Kiss H, Begtrup H, Ball M, Iatrop M: Environmental tobacco smoke in real life situations. Proc Fourth Int Conf Indoor Air Quality and Climate 1987; 137-141.
- NIOSH: Manual of Analytical Methods, 3rd Ed. Method 3501. DHHS Pub. No. 84-100. Cincinnati: NIOSH, 1984.
- Dally KA, Haarhaus LP, Woodbury MA, Kanarek MS: Formaldehyde exposure in nonoccupational environments. *Arch Environ Health* 1981; 36:277-284.
- Ritchie I, Lehnen RG: An analysis of formaldehyde concentrations in mobile and conventional homes. *J Enviro Health* 1983; 47:300-305.
- Broder I, Corey F, Cole P, Mintz S, Pa ML, Nethercot J: Health status of residents in homes insulated with urea-formaldehyde foam. In: Proceedings APC Specialty Conference—Indoor Air Quality in Cold Climates: Hazards and Abatement Measures, 1986; 153-156.
- Otoole JH, Dossing M: Formaldehyde induced symptoms in day care centers. *Ind Hyg Assoc J* 1982; 43:366-370.
- Ritchie DM, Lehnen RG: Formaldehyde-related health complaints of residents in mobile and conventional homes. *Am J Public Health* 1987; 77:323-328.
- Hendrick DJ, Rander RJ, Lane DJ, Morris MJ: Formaldehyde asthma: challenge exposure levels and fate after five years. *JOM* 1982; 24:893-897.
- Nordinas H, Keskinen H, Tuppurainen M: Formaldehyde asthma—rare or overlooked. *J Allergy Clin Immunol* 1983; 73:91-99.
- Office of Pesticides and Toxic Substances, USEPA: Assessment of health risks to garment workers and certain home residents from exposure to formaldehyde. Washington, DC: USEPA, 1987.

2023380488

---

NOTICE

This material may be  
protected by copyright  
law (Title 17 U.S. Code).

# Residential Formaldehyde

## *Increased exposure levels aggravate adverse health effects*

Thad Godish, Ph.D.

---

### Abstract

Residents of mobile homes and conventional homes with particleboard subflooring present were studied to determine the relationship between residential formaldehyde levels and the severity of a variety of reported health symptoms. Formaldehyde concentrations were measured concurrently with occupant ratings of symptom severity. Levels were relatively low with median values in mobile homes of 0.12 ppm and 0.07 ppm in conventional homes with particleboard subflooring. Significant relationships were observed for 16 symptoms including eye irritation, dry/sore throat, runny nose, cough, sinus irritation, sinus infection, headaches, fatigue, depression, difficulty sleeping, rashes, bloody nose, nausea, diarrhea, chest pain and abdominal pain. A significant independent effect of formaldehyde on symptom severity was observed even after adjusting for potential confounding variables such as the presence of a smoker in the household, individual age and season of the year.

Formaldehyde is a ubiquitous contaminant of indoor air, particularly in residential environments where concentrations often exceed ambient (outdoor) values by one or more orders of magnitude. Elevated levels have been reported for mobile homes, for conventional homes with particleboard subflooring, for urea-formaldehyde foam-insulated homes, and for a variety of conventional homes utilizing urea-formaldehyde-based pressed wood products and finish coatings (1,2,3,4,5).

Formaldehyde is a potent mucous membrane irritant (6), is known to cause asthma (7,8), and has been implicated as a human carcinogen (9,10). Formaldehyde indoor air contamination has been the focus of numerous investigations of building-related health complaints (1,2,3) and several prospective (11,12,13) and retrospective (14,15) epidemiological studies.

The study reported here is based on a statistical analysis of data collected in investigations of homeowner/occupant complaints and requests for public health assistance. It was primarily designed to determine whether significant relationships exist between residential formaldehyde exposure levels and the severity of subjective symptoms. It is based on a study protocol of formaldehyde sampling of two source residence types (mobile homes and conventional houses with particleboard subflooring) with concurrent administration of a health survey questionnaire and occupant ranking of symptom severity.

Thad Godish, Ph.D., Director, Indoor Air Quality Research Laboratory, Ball State University, Muncie, IN 47306.

### Methods

Data which served as the basis of this study were collected over a five-year period in a cooperative program of residential investigations, formaldehyde air testing and health surveys conducted by the Indoor Air Quality Research Laboratory, Ball State University, the Industrial Hygiene Division of the Indiana State Board of Health and seven county health departments.

Study participants were drawn from households requesting air testing assistance from county health departments. Assistance was provided when requests were based on health concerns associated

with indoor air quality. Formaldehyde sampling, source identification, and the administration of a health survey questionnaire were conducted by trained county environmental health personnel.

Prior to each field investigation, principal occupants were advised to maintain closure conditions (windows and doors) prior to (for a minimum of 12 hours) and during the scheduled air testing period.

All formaldehyde sampling was conducted with bubbler samplers for a duration of 90 minutes at a sampling rate of 1 liter/minute. Samples were analyzed by the Industrial Hygiene Division of the Indiana State Board of Health using the modified NIOSH chromotropic acid method (16). All samples were collected in a 15 ml solution of 1% sodium bisulfite. Samples were shipped or transported directly to the analytical laboratory in Fisher nalgene polyethylene bottles. A minimum of one formaldehyde sample was collected in the major living area of each residence.

Each residence was inspected to identify major formaldehyde sources. A health survey questionnaire was administered to only those occupants present. At the time of the interview, neither the respondent nor the interviewer knew what the formaldehyde concentration was in the house.

Each occupant present (12 years or older) was asked to rate the severity of 22

---

Table 1  
Residential Formaldehyde Levels in Study Residences

Type of residence	No.	Median	Formaldehyde Level Number (percent) in each ppm range:				
			<0.10	0.10-0.19	0.20-0.29	0.30-0.40	>0.40
Mobile homes	82	0.120 ppm	25 (40.3)	19 (30.6)	12 (19.4)	2 (3.2)	4 (6.5)
Conventional (Particleboard Subflooring)	52	0.070 ppm	35 (67.3)	15 (28.8)	1 (1.9)	0 (0)	1 (1.9)
Mobile and Conventional (Particleboard Subflooring)	14	.090 ppm	60 (52.6)	34 (29.8)	13 (11.4)	2 (1.8)	5 (4.4)

health symptoms on a scale from zero to four, adapted from Evans, et al (16): (0) no symptoms, (1) slight-symptoms present without thinking about it but not annoying, (2) moderate-symptoms present without thinking about it but is annoying, (3) severe-symptoms limit activity, and (4) very severe-symptoms are incapacitating, resulting in a discontinuation of ongoing activity. Occupants were further interviewed to obtain demographic data and other information.

Data were analyzed by the application of standard statistical procedures utilizing the statistical package for the social sciences (SPSSX). Chi-square analysis was applied in the initial data analysis to determine interrelationships between demographic characteristics, house factors, season of the year, residential formaldehyde levels and the frequency of symptoms.

Spearman's rank correlation test was applied to the formaldehyde level and individual symptom severity ratings of residents. Multiple regression analyses were applied to symptom severity indices developed for three symptom groupings including those affecting the lower respiratory system (cough, wheezing, difficulty breathing), mucous membranes (eye irritation, eye infections, sinus irritation, sinus infection, dry/sore throat, runny nose) and central nervous system (headaches, dizziness, depression, difficulty sleeping, memory loss, sensations in extremities). Indices were developed by summing severity ratings in a symptom grouping for each respondent.

Formaldehyde levels were log transformed (base 10) to normalize the distribution of this variable for inclusion into the multiple regression model as an independent variable. Potential confounding factors, such as age, season and the presence of a smoker in the household, were included in the multiple regression analysis to evaluate relationships between symptom severity and residential formaldehyde levels. A probability level of 0.05 was considered significant.

## Results

Formaldehyde levels in the study population of 61 mobile homes and 52 conventional homes with particleboard subflooring are summarized in Table 1. In both housing groups, median formaldehyde levels were relatively low; 0.12 ppm in mobile homes; 0.07 ppm in conventional houses with particleboard subflooring. In the former, 40% of formaldehyde values were less than 0.10 ppm, the ASHRAE recommended guideline for indoor air. In the latter, 67% of formaldehyde values were below the ASHRAE guideline.

Chi-square analyses applied to determine the inter-relationships between demo-

Table 2

Spearman's Rank Order Correlation between Residential Formaldehyde Levels and Individual Symptom Severity Ratings

	Mobile Homes	# with symptom	PB sub-flooring	# with symptom	Pooled data	# with symptom
Symptom	r =		r =		r =	
Eye Irritation	0.2097**	46	0.1392*	30	0.2115**	76
Eye Infection	-0.0694	14	-0.0237	9	-0.0365	23
Dry, Sore Throat	0.2849**	60	0.2262**	48	0.2685**	106
Runny Nose	0.1785**	65	0.1679*	54	0.1881**	119
Cough	0.2185**	60	0.0662	51	0.1419**	111
Sinus Irritation	0.3317**	69	0.1834*	55	0.2595**	124
Sinus Infection	0.2848**	31	0.0563	20	0.1832**	51
Difficulty Breathing	0.0761	46	0.0230	32	0.0842	78
Headaches	0.1688**	61	0.1865**	44	0.1988**	105
Dizziness	0.0356	29	0.0719	20	0.0712	49
Fatigue	0.3352**	61	0.1671*	41	0.2745**	102
Depression	0.2036**	36	0.1079	23	0.1862**	59
Difficulty Sleeping	0.1638**	58	0.1752*	33	0.2089**	92
Rashes	0.3111**	24	0.0622	19	0.1931**	43
Bloody Nose	0.1254	11	0.1340*	10	0.1305*	21
Nausea	0.1130	19	0.1768*	15	0.1454**	34
Diarrhea	0.0681	26	0.0721	10	0.1217*	36
Chest Pain	0.0690	27	0.2276**	20	0.1764**	47
Abdominal Pain	0.1014	20	0.1593*	16	0.1426**	36
Menstrual Problems <sup>a</sup>	0.0668	6	0.0560	7	0.0603	13
Wheezing	0.0911	26	-0.0488	25	0.0335	51
Asthmatic Attacks	0.0774	9	-0.0087	15	0.0207	26
Extremity Sensations	-0.0958	7	-0.0395	8	-0.0623	15
Memory Loss	-0.1108	9	-0.0850	7	-0.0768	16
Muscular Pain	-0.0365	9	0.0503	8	0.0234	17

\* p < 0.05

\*\* p < 0.01

<sup>a</sup> Women between 12 and 45 years of age.

graphic characteristics, season of the year, residential formaldehyde levels and symptom frequency revealed that females had a significantly higher frequency of headaches, fatigue, depression, difficulty in sleeping and abdominal pain than men in the study.

Occupants of homes where a smoker resided reported significantly higher frequencies of eye irritation, dry/sore throat, runny nose, cough, headache, depression, sinus infection, dizziness, fatigue, difficulty in sleeping and chest pain than occupants of homes in which no smokers resided.

When the effect of season was evaluated,

dry/sore throat, runny nose, sinus infections, headaches, dizziness, nauses and chest pain were more frequent in the spring months. Higher rates of runny nose, fatigue and depression were observed in the winter. High rates of fatigue and depression were also observed during the fall. No significant relationships were observed between symptom frequency and the type of cooking stove (gas vs. electric) or type of heating system (gas vs. electric).

Results of Spearman's rank correlation between individual symptom severity ratings and formaldehyde levels are summarized in Table 2. Significant relationships between formaldehyde levels and

2023380490

## Significance of exposure to benzene and other toxic compounds through environmental tobacco smoke\*

F. Adlikofer<sup>1</sup>, G. Scherer<sup>1</sup>, C. Coaze<sup>1</sup>, J. Angerer<sup>2</sup>, and G. Lehnert<sup>2</sup>

<sup>1</sup> Analytisch-biologisches Forschungslabor, Prof. Dr. F. Adlikofer, Goethestrasse 20, D-8000 München 2, Federal Republic of Germany

<sup>2</sup> Institut für Arbeits- und Sozialmedizin, Schillersstrasse 25, D-8520 Erlangen, Federal Republic of Germany

Received 28 June 1990 / Accepted 29 June 1990

**Summary.** In order to assess the uptake of benzene from environmental tobacco smoke (ETS) and to estimate its contribution to the total body burden of benzene observed in non-smokers, two experimental studies have been conducted. Controlled exposure to high levels of ETS equivalent to 10 ppm CO for 9 h and 20 ppm for 8 h resulted in a nonsignificant increase in blood benzene levels and a significant increase in exhaled CO, COHb and cotinine in serum and urine. The slightly rising blood concentration of benzene following experimental ETS exposure was paralleled by an increased exhalation of benzene and aromatic hydrocarbons and in contrast to blood levels, this increase was significant. The blood levels of benzene obtained during exposure were comparable to those observed at the time of admission to the laboratory, when biomarkers of ETS uptake, e.g. cotinine in serum and urine, were at the limit of detection, thus demonstrating that these background levels were not from ETS exposure. No difference in the urinary excretion of phenol, the main metabolite of benzene, was found during the experimental periods. The background levels of urinary phenol in unexposed nonsmokers were rather high, demonstrating that phenol excreted in urine must be formed from several endogenous and exogenous precursors. In the light of our findings it is highly questionable whether exposure to benzene from ETS under real life conditions poses a cancerogenic risk to the general population, which is measurable today or in the future by toxicological or epidemiological methods.

**Key words:** ETS – benzene – biomonitoring – risk evaluation

### Introduction

Benzene is a human carcinogen that is ubiquitously found in the environment (WHO 1987; IARC 1988; Gesundheitsschädliche Arbeitsstoffe 1989). Foremost among its many sources is automobile emissions. In the Federal Republic of Germany approximately 42 000 tons of this pollutant are estimated to be emitted into the environment on an annual basis. Additional sources, including petrochemical industry emissions, industrial and home heating systems annually contribute another 4000 tons of this pollutant (BUA-Stoffbericht 1988).

Benzene and other aromatic hydrocarbons are formed during the burning of tobacco (Hoffmann et al. 1989). While the yield of these hydrocarbons in mainstream smoke that is inhaled by the smoker can be selectively reduced by charcoal filters (Hoffmann et al. 1989) and by ventilation (Kiefer 1978), the concentration in sidestream smoke, the major portion of environmental tobacco smoke (ETS), cannot at present, be effectively diminished.

Sidestream smoke of a burning cigarette emits between 120 µg and 480 µg benzene into the ambient air (IARC 1986). Since approximately 150 billion cigarettes are consumed in this country on an annual basis, ETS might be expected to be responsible for as much as 0.1% of the national burden of this pollutant. In contrast, however, to the benzene released from industrial and automobile sources, which is generally distributed in the outdoor air before it penetrates into offices and dwellings, benzene from cigarette smoke is mainly emitted directly into the indoor environment.

In order to assess the uptake of benzene and other aromatic hydrocarbons through passive smoking, we have conducted two experimental studies with nonsmokers. In the first study, eight nonsmokers were exposed to ETS equivalent to a CO concentration of about 10 ppm for 9 h. Blood samples and exhaled air were examined for benzene and other biomarkers before, during and after exposure to ETS. In the second study, five nonsmokers were exposed to the gaseous phase of ETS equivalent to

\* Dedicated to Professor Dr. Dietrich Schmahl on the occasion of his 65th birthday

**Abbreviations:** ETS, environmental tobacco smoke; BTX, benzene/ethylbenzene/toluene/m- and p-xylene/o-xylene

**Offprint requests to:** F. Adlikofer

2023380491

about 20 ppm CO on one day and whole ETS at the same concentration on another day. Blood samples were analysed for benzene and for markers of ETS uptake. Urine samples were evaluated for the presence of metabolites and for mutagenicity.

### Materials and methods

In study 1, eight healthy male nonsmokers (aged 21–40 years) and three smokers took part in the investigation. They reported to the laboratory on Monday evening and remained there until the termination of the study on Wednesday morning. In study 2, five healthy male nonsmokers aged 23–29 years and five smokers stayed in the laboratory from Saturday evening until the following Friday morning. All subjects completed a questionnaire on socioeconomic and life-style factors as well as their smoking habit, ETS exposure and the meals they had consumed during the preceding 48 h. During both studies, the subjects ate a defined diet low in polycyclic hydrocarbons (Marun et al. 1989). This diet was identical in quality and quantity on each day of the investigation. Smokers were not allowed to smoke in the laboratory except during the experimental smoking periods.

**Protocol: study 1.** On the day after admission to the laboratory (Tuesday), the eight nonsmokers spent 9 h (10.30 a.m. to 7.30 p.m.) in a furnished room (45 m<sup>3</sup>) during which time three smokers smoked a total of 102 cigarettes. Smoking frequency was controlled so that the CO concentration in the room air was maintained at around 10 ppm. The subjects were allowed to leave the room only for sampling of blood and exhaled air. Blood and exhaled air were sampled according to the time schedule indicated in Fig. 1. Two urine fractions were collected: the first sampling period lasted from the evening when the subjects arrived at the laboratory until the next morning (about 12 h), while the second urine fraction collection continued until the morning of the next day (24 h).

**Protocol: study 2.** On the first day of the study (Sunday), the nonsmokers remained in the laboratory to guarantee that they had no ETS exposure. On the second day (Monday), they spent 8 h (8.30 a.m. to 4.40 p.m.) in a furnished room (45 m<sup>3</sup>) without any ETS exposure. On the third day (Tuesday), they were exposed for 8 h (8.30 a.m. to 4.30 p.m.) to the gaseous phase of ETS produced by five smokers in the same room smoking 120 cigarettes. The nonsmoking subjects wore masks equipped with filters (Sekur Polimask OC; Filter classes P1 and P2, Prelli, FRG), which removed more than 99% of the particulate mass from the inspired air. On the fourth day (Wednesday), subjects remained in the room, as on the second day, without exposure to ETS. On the fifth day (Thursday), the nonsmokers were exposed to whole ETS for 8 h (8.30 a.m. to 4.30 p.m.) sitting in the room without masks. The smokers generating the ETS in the room smoked the same number of cigarettes according to the same time schedule on the third day.

Blood and exhaled air were sampled at the times indicated in Fig. 2. Urine was collected at intervals of 24 h beginning at 8.00 a.m. on each day. The analyses outlined in the present paper are based only on data obtained from the passive smokers.

**Air monitoring.** For study 1 and study 2, air sampling tubes were installed at breathing height of a seated person at the end of the room opposite to where the smokers sat. Carbon monoxide was measured continuously by an infrared CO monitor UNOR 6N (Fa. Maihak, Hamburg, FRG). Nitrogen oxides (NO/NO<sub>2</sub>) were detected by a chemiluminescence monitor using a nitrogen oxide analyser, model 8840 (Monitor Labs Inc., USA). Air nicotine was determined according to the method of Ogden (1986). The alkaloid was absorbed onto XAD-4 resin with an air flow rate of 1 l/min. Sampling times were 4 h on the sham-exposure days and 2 h on the exposure days. Formaldehyde was absorbed on Sep-PAK C<sub>18</sub> (Waters Associated,

Milford, Mass., USA) coated with 2,4-dinitrophenylhydrazine and determined by HPLC (Kuwata et al. 1983). Flow rates and sampling times were the same as those for nicotine. Respirable particles were determined gravimetrically according to the method of Oldaker et al. (1990). The sampling flow rate was 1.6 l/min. Sampling periods were similar to those for nicotine. In study 2, polycyclic aromatic hydrocarbons were detected according to the method of Grimmer et al. (1987). The sampling period was 8 h. The filter system consisted of a siliconized glass-fibre filter and a Pore Pak PS filter for sampling particles and semi-volatiles, respectively.

Benzene as well as ethylbenzene, toluene, *m*- and *p*-xylene and *o*-xylene (BTX) were measured by a modification of the NIOSH method (NIOSH 1984) using gaschromatography/mass spectrometry after absorption on charcoal tubes (Dräger AG, Lübeck, FRG) and elution with carbon disulfide (NIOSH 1984). Sampling time ranged from 10 min to 60 min with flow rates ranging from 0.5 l/min to 2 l/min.

Except for nicotine, the same compounds were also measured applying the same sampling conditions on the sham-exposure days. Carbon monoxide and nitrogen oxides were determined off-line in air samples collected in 5-l plastic air bags by means of a hand pump.

**Biomonitoring.** Carboxyhaemoglobin (COHb) was determined spectrophotometrically on fresh blood samples with an IL 282 CO oximeter (IL 282, Instrumentation Laboratories Ltd., USA). Nicotine was determined in plasma and urine by gas chromatography (Hengen and Hengen 1978) and cotinine in plasma by radioimmunoassay (Langone et al. 1973). Thioethers were measured in urine by quantifying the sulphhydryl groups with Ellman's reagent after alkaline hydrolysis of the thioether bonds according to the method of Heinonen et al. (1983) as modified according to Aringer and Lundström (1988).

Urinary mutagenicity was determined as previously described using the *Salmonella typhimurium*/mammalian microsome assay. Strain TA98 was utilized as the tester strain (Scherer et al. 1987). The following modifications to this method were employed. 500 ml urine was used for extraction and loaded XAD-2 columns were washed with 100 ml methylene chloride. The evaporated eluate was dissolved in 0.75 ml dimethylsulphoxide. The test was performed with 10-, 25-, 50-, and 75-µl aliquots of this extract. These aliquots were equivalent to 6.7, 16.7, 33.3, and 50 ml urine, respectively. The spontaneous mutation rate in the presence of S9 mix (supernatant derived from the centrifugation of the liver homoginate at 9000 g) was 35–55 revertants/plate.

Benzene in 2-ml blood samples was determined by dynamic head-space chromatography and flame ionisation detection as described elsewhere (Angerer et al. 1990). Briefly, the volatile organic compounds are purged from the incubated blood vials (30 min at 60° C) by nitrogen and collected at –30° C on a Tenax absorption tube (3 mg). Transfer from the trap to the capillary column (DB 1301) of the gas chromatograph (Sichromat 1–4 Siemens, FRG) was made by flash heating (300° C) of the trap. The calibration was carried out with spiked animal blood samples in the range of 0.2–10 µg benzene/l.

Carbon monoxide in exhaled air was determined by a UNOR 6 N CO monitor (Maihak, FRG). Breath sampling for BTX measurements was performed according to the procedure of Pellizzetti et al. (1988). Briefly, the subjects inhaled purified air from a 60-l Tedlar bag and exhaled it via a breathing valve into a second 60-l Tedlar bag. The procedure lasted about 10 min until about 40 l exhalate was sampled. A 30-l sample of the exhaled air was pumped through a charcoal tube (Dräger AG, Lübeck, FRG) at a flow rate of 1.5–2 l/min and BTX was determined as described for ambient air samples.

**Statistical analysis.** The *t*-test for paired samples (exposed versus non-exposed) was applied. For blood and plasma parameters, the level in the respective morning sample before start of the exposure was used as the non-exposed reference value. In the case of urine parameters, the level in the urine fraction before exposure was used as the non-exposed reference value.

2023380492

## Results

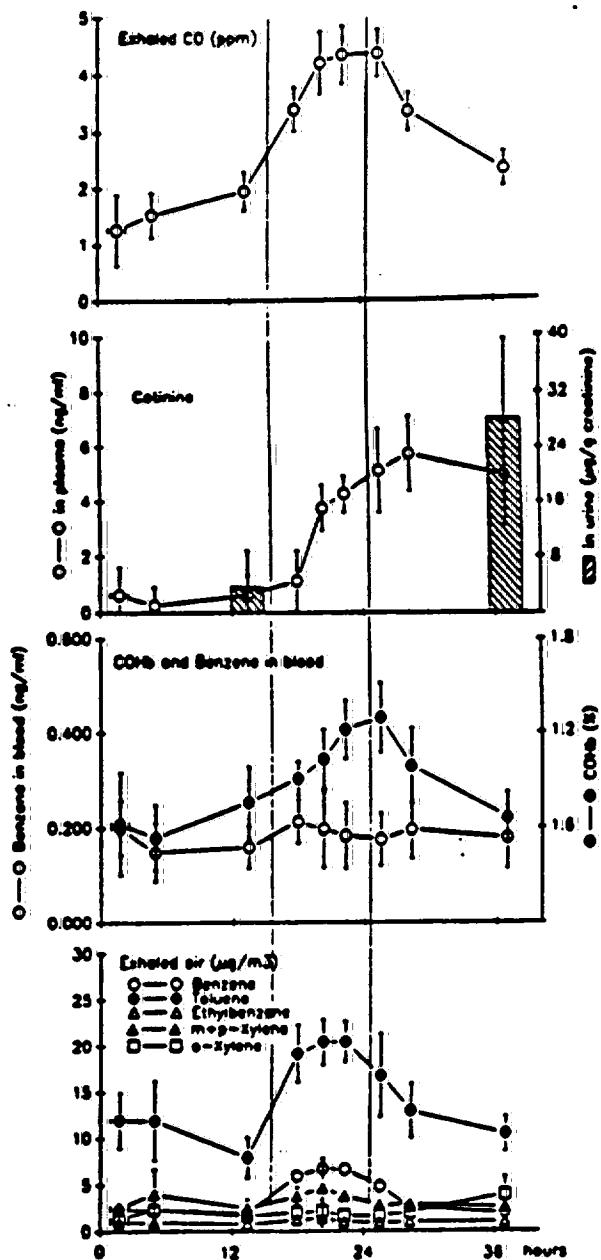
The time-weighted average concentrations of gas-phase components as well as of particulate matter to which the nonsmokers were exposed in the first study are summarized in Table 1. A strong wind prevailed during the study period, ventilating the experimental room through its poorly sealed windows. This could account for the low background levels of measured constituents that were observed during study 1 as well as the high smoking rate, which was necessary to reach and maintain a constant ETS concentration equivalent to about 10 ppm CO.

The biomonitoring results for the nonsmokers in this study are shown in Fig. 1. The high COHb levels and the elevated amounts of CO in the exhaled air as well as the increased concentrations of cotinine in serum and urine indicate that ETS exposure was high and not representative of what can be expected in real-life settings. We found a slight increase in the blood benzene concentrations at the beginning of the exposure period with the maximum level not exceeding that observed in the subjects upon admission to the laboratory. The observed benzene level noted upon admission to the laboratory could not be due to ETS exposure since cotinine in both serum and urine was at the level of detection and therefore not indicative of recent ETS exposure. Upon ETS exposure in the laboratory, the increase in blood concentration of benzene was paralleled by an enhanced exhalation of benzene and other aromatic hydrocarbons in expired air. In contrast to the blood levels of benzene, this increase in exhaled benzene was significant.

In order to maximize the probability that the inhalation of ETS compounds leads to an increase in biological markers in body fluids above the detection limit, a second experiment under even more extreme ETS exposure conditions was carried out. The time-weighted average concentrations of ETS components the nonsmokers were ex-

**Table 1.** Concentration of gaseous-phase components and particulate matter in an experimental room before, during and after smoking 102 cigarettes in 9 h. Data are time-weighted averages: 16 h before, 9 h during and 8 h after smoking (study 1)

Component	Content		
	Before	During	After
		smoking	
<b>Gaseous phase</b>			
CO (ppm)	0.4	9.5	0.6
NO <sub>x</sub> (ppm)	24.7	187.3	17.8
Formaldehyde ( $\mu\text{g}/\text{m}^3$ )	23	180	40
Acetaldehyde ( $\mu\text{g}/\text{m}^3$ )	19	633	17
Nicotine ( $\mu\text{g}/\text{m}^3$ )	0.6	63.6	6.8
Benzene ( $\mu\text{g}/\text{m}^3$ )	4.8	60.1	3.0
Toluene ( $\mu\text{g}/\text{m}^3$ )	15.7	286.2	17.3
Ethylbenzene ( $\mu\text{g}/\text{m}^3$ )	3.1	18.1	2.0
m,p-Xylene ( $\mu\text{g}/\text{m}^3$ )	6.8	53.8	5.1
p-Xylene ( $\mu\text{g}/\text{m}^3$ )	8.0	24.8	7.0
<b>Particulate matter</b>			
Respirable suspended particles ( $\mu\text{g}/\text{m}^3$ )	25	1803	52



**Fig. 1.** Biomonitoring for study 1: results are presented as means with standard deviation bars for five nonsmokers before, during and after experimental environmental tobacco smoke exposure. The dotted lines indicate the 9-h ETS exposure session.

posed to in this study are summarized in Table 2. A smoking-related increase is clearly seen in all parameters measured. Some particulate matter constituents (respirable suspended particles, benzo[a]pyrene, benzo[e]pyrene, benz[a]anthracene) show higher concentrations on the fifth day than on the third day even though the same number of cigarettes was smoked. The reasons for this are unclear. The poor ventilation conditions in the room on this day with high temperatures (28–35°C) and a high relative humidity could have affected the measurements.

2023380493

Table 2. Air monitoring in the experimental room. Data are time-weighted averages for the 9-h exposure sessions.

Component		Yield on days			
		2 -	3 (120)*	4 -	5 (120)*
<b>Gaseous phase</b>					
CO	(ppm)	1.4	24	2.0	24
NO <sub>x</sub>	(ppb)	38	422	56	449
Formaldehyde	( $\mu\text{g m}^{-3}$ )	3	48	3	49
Acetaldehyde	( $\mu\text{g m}^{-3}$ )	290	1450	85	1390
Propionaldehyde	( $\mu\text{g m}^{-3}$ )	15	120	14	120
Nicotine	( $\mu\text{g m}^{-3}$ )	4	71	6	71
Benzene	( $\mu\text{g m}^{-3}$ )	8	190	12	206
Phenanthrene	( $\text{ng m}^{-3}$ )	138	154	nd	258
Pyrene	( $\text{ng m}^{-3}$ )	29	24	nd	25
<b>Particulate matter<sup>b</sup></b>					
RSP	( $\mu\text{g m}^{-3}$ )	77	3181	78	4091
BaP	( $\text{ng m}^{-3}$ )	0.2	21.5	0.3	26.7
BeP	( $\text{ng m}^{-3}$ )	0.8	21.5	0.8	24.9
Coronene	( $\text{ng m}^{-3}$ )	0.1	2.8	0.6	2.2
Anthanthrene	( $\text{ng m}^{-3}$ )	0.06	3.9	0.07	3.1
Benzofluoranthenes (b-j-k)	( $\text{ng m}^{-3}$ )	2.1	52.3	1.7	55.3
Chrysene	( $\text{ng m}^{-3}$ )	1.8	54.2	1.5	70.5
BaA	( $\text{ng m}^{-3}$ )	2.1	18.9	1.1	26.2
Phenanthrene	( $\text{ng m}^{-3}$ )	3.7	6.8	1.8	7.4
Pyrene	( $\text{ng m}^{-3}$ )	0.6	17.6	0.7	20.5
NNN	( $\text{ng m}^{-3}$ )	1	4	1	5
NNK	( $\text{ng m}^{-3}$ )	1	9	1	6

\* On days 3 and 5 120 cigarettes were smoked in 9 h.

<sup>b</sup> RSP, respirable suspended particles; BaP, benzo[a]pyrene; BeP, benzo[e]pyrene; BaA, benz[a]anthracene; NNK, 4-(N-nitroso-methylamino)-1-(3-pyridyl)-1-butaneone; NNN, N-nitrosoborane.

Phenanthrene and pyrene were found in both particulate matter and the gas phase of ETS (Table 2). They are more abundant in the gas phase of ETS, although the background levels for both substances were rather high, particularly for the volatile component. Concomitantly, the background levels for NO<sub>x</sub> and acetaldehyde were also high. This is certainly due to the environmental air pollution around the office building, which is located in the center of the city of Munich.

The biomonitoring results for the nonsmokers in this study are shown in Fig. 2a, b. COHb, nicotine in plasma and urine and thioether excretion in urine increased after exposure to both gas phase and whole ETS. The rise in urinary thioether excretion due to both exposures is only of borderline significance, whereas the increases in COHb and nicotine are significant when compared to the pre-exposure levels. After exposure to whole ETS, urinary mutagenicity seemed to be slightly elevated. However, a similar elevation was measured on the second day, before there was any experimental exposure to ETS (Fig. 2a). There was a slight increase in the benzene levels in blood after exposure to gas-phase and whole ETS, but the benzene concentrations obtained were no higher than those measured before exposure during the day after admission to the laboratory (Fig. 2b). These levels were not caused by ETS exposure, because the cotinine serum and urine concentrations were lower than 2 ng/ml and 2  $\mu\text{g/l}$ ,

24 h, respectively. No difference in the urinary excretions of phenol, the main metabolite of benzene, was found during the experimental periods. The background levels of urinary phenol were rather high, suggesting that phenol excreted in urine must be formed from several endogenous and exogenous precursors. The same applies to *m*- and *p*-cresol.

Our findings are in line with the fact that exposure agents such as CO, nicotine, benzene and many electrophiles (giving rise to thioether formation) are constituents of the gas phase of ETS since they are found following exposure to gas phase alone as well as to whole ETS.

## Discussion

As seen in Table 3, the uptake of particulate-phase constituents by passive smoking is up to three orders of magnitude lower whereas the uptake of gas phase compounds is only less than one order of magnitude lower than with active smoking. Therefore, it is evident that for passive smoking, exposure to the gas phase is more relevant than exposure to the particulate phase of ETS. The increases in COHb, nicotine, cotinine in plasma, and in the urinary excretion of nicotine, cotinine and thioethers are all due to the uptake of gaseous-phase compounds. This, as well as the fact that urinary mutagenicity did not change during exposure to ETS, has been discussed in a previous paper (Scherer et al. 1990).

In this study, we have investigated the uptake of a special gaseous-phase compound, i.e. benzene, through passive smoking. Under the exposure conditions chosen, an increased benzene level in blood as well as in exhaled air was found. Phenol, the major metabolite of benzene (Snyder 1987; WHO 1987; Norpoth 1989), was not increased in the urine of nonsmokers after ETS exposure. The high urinary excretion of phenol indicates that there are many precursors of exogenous and endogenous origin and that the contribution by benzene due to ETS exposure even under extreme conditions is not significant.

The ETS exposure concentrations equivalent to 10 ppm CO, as used in the first study, resulted in urinary cotinine levels that were about twice as high as had been measured in 20% of two free living populations of nonsmokers reporting the highest levels of ETS exposure (Becher et al. 1987; Haley 1989). If this comparison is made, it has to be considered that in our study, after having reached a steady state within several exposure days, the urinary cotinine excretion would have been much higher.

Although the data of Wallace (1989) indicate such a possibility, it remains doubtful whether an increased benzene concentration in blood or in exhaled air, as observed after an ETS exposure, can be found under real-life conditions. Median levels of benzene in 200 homes without smokers in the USA were 7  $\mu\text{g m}^{-3}$ , while in 300 homes with one or more smokers 10.5  $\mu\text{g m}^{-3}$  were measured (Wallace 1989). In a study of 500 homes in the Federal Republic of Germany, median values of 6.5  $\mu\text{g m}^{-3}$  were found in nonsmoking homes and 11  $\mu\text{g m}^{-3}$  in smoking homes (Krause et al. 1987). In two other studies, no dif-

2023380494

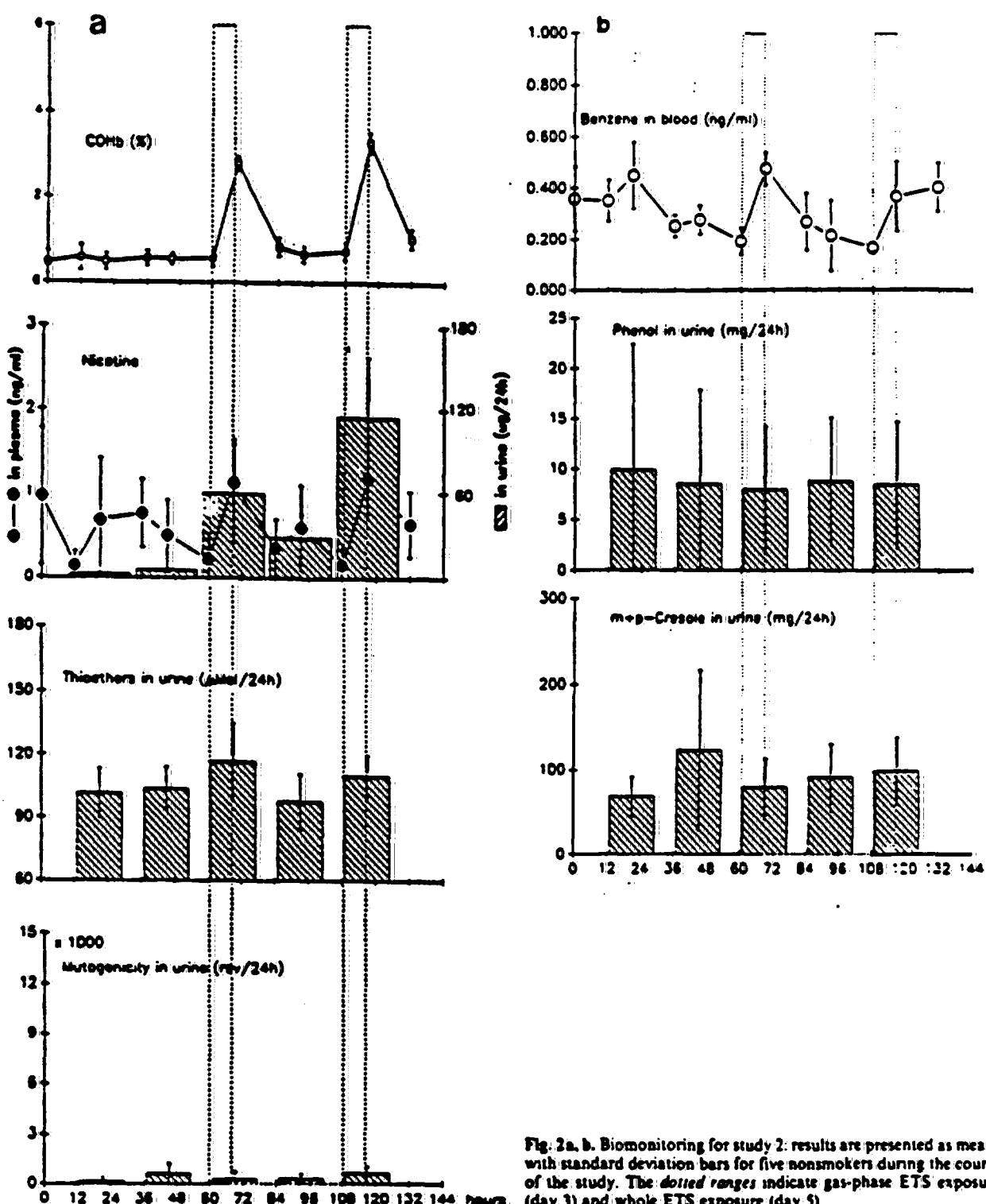


Fig. 2a, b. Biomonitoring for study 2: results are presented as means with standard deviation bars for five nonsmokers during the course of the study. The dotted ranges indicate gas-phase ETS exposure (day 3) and whole ETS exposure (day 5)

ference was found in homes (Proctor et al. 1990a) and office buildings (Proctor et al. 1989) with and without smokers. If benzene intake, based on a 24 h respiratory volume of 20 m<sup>3</sup> at rest, will be 10 µg per day for each 1 µg per m<sup>3</sup> in the air, ETS exposure may contribute

45 µg benzene per day at the highest. This is about 10% of the daily benzene intake in a nonsmoker and about 3% in a smoker (WHO 1987).

As can be seen from Table 4, benzene concentrations in indoor air of an office building located in the center of

2023380495

Table 3. Estimated dose ratio between smoking (20 cigarettes/day) and passive smoking (8 h/day)<sup>a</sup>

Tobacco smoke constituents	Smoking <sup>b</sup> (S)	Passive smoking <sup>c</sup> (PS)	Dose ratio (S/PS)
<b>Gaseous phase</b>			
CO	(mg) 40 - 400	14.4 - 96	2.7 - 4.2
Formaldehyde	(mg) 0.4 - 1.8	0.08 - 0.4	4 - 5
Volatile nitroamines	( $\mu$ g) 0.05 - 1.0	0.03 - 0.4	1.5 - 2.5
<b>Particulate matter</b>			
Particles	(mg) 75 - 300	0.024 - 0.24	1250 - 3000
Nicotine <sup>d</sup>	(mg) 7.5 - 30	0.08 - 0.4	75 - 90
Benzo[a]pyrene	( $\mu$ g) 0.15 - 0.75	0.001 - 0.011	70 - 150
Cadmium	( $\mu$ g) 1.5	0.001 - 0.014	110 - 1500
Tobacco-specific nitroamines	( $\mu$ g) 4.5 - 45	0.002 - 0.010	2300 - 4500

<sup>a</sup> Data are compiled from Sterling and Dimich (1982), IARC (1986), Surgeon General (1986), Khus and Begotter (1987).

<sup>b</sup> Assumed deposition rate for particulate matter: 75% (Hinds et al. 1983)

<sup>c</sup> Assumed breathing volume: 0.5 m<sup>3</sup>/h; assumed deposition rate for particulate matter: 11% (Hiller et al. 1982)

<sup>d</sup> Nicotine is particle-bound in main-stream and a gas-phase constituent in ETS (Eudy et al. 1986).

Table 4. Benzene, toluene and xylylene measurements in an office building in the center of Munich and in a minibus driving through the city of Munich

Date/time	Location/events	Amount ( $\mu$ g/m <sup>3</sup> )				
		Benzene	Toluene	Ethylbenzene	m,p-Xylene	<i>o</i> -Xylene
16 Oct. 1989						
19.30-19.40	Office building	38	195	16	54	16
21.30-21.40	Office building	32	103	16	49	16
23.20-23.30	Office building	16	38	5	22	5
17 Oct. 1989						
08.35-08.50	Driving into the center, dense traffic	68	141	26	90	34
09.30-09.40	Driving near the main station, passing a tunnel, stop and go	102	198	38	128	45
10.00-10.10	Driving through the center, stop and go	102	211	45	147	51
10.30-10.40	Driving back to the office building, stop and go	64	153	32	96	38
11.00-11.10	Driving out of the center, dense traffic	58	131	26	83	32
11.14-11.20	Stop at a gasoline station, refuelling	1000	3400	700	2600	1000
11.30-11.40	Parking about 30 m away from the gasoline station, light traffic passing	32	102	26	83	32
12.00-12.10	Driving back to the center, dense traffic	64	153	26	109	38
12.30-12.40	Driving in the center, dense traffic	141	275	51	166	64
13.00-13.10	Stop close to a park in the center, sampling outside the car, 20 m away from the road	39	83	19	58	19
13.30-13.40	Some location but sampling in the car, parking on the road	45	96	19	58	19
14.00-14.10	Driving in the center, light traffic	38	83	19	58	19
14.30-14.40	Parking in front of the office building, dense traffic passing	96	198	38	134	45
15.00-15.10	Driving in the center, dense traffic	70	147	32	102	38
15.17-15.20	Stop at a gasoline station, refuelling	13000	28500	3700	13600	4600
15.30-15.40	Parking 50 m away from the gasoline station, light traffic passing	96	377	102	441	166
16.00-16.10	Parking on a road with dense traffic passing	51	128	32	109	38
16.30-16.40	Parking in front of the office building, normal traffic passing	26	96	32	128	58
17.00-17.10	Driving close to the main station, dense traffic	83	173	38	147	58
17.30-17.40	Office building	19	58	13	32	13

the city of Munich amount to up to 38  $\mu$ g/m<sup>3</sup>, while in a small bus occupied with eight subjects and driven through the city, values of up to 141  $\mu$ g/m<sup>3</sup> can be measured. Additionally, during stops at two different petrol stations, 1000 and 13000  $\mu$ g/m<sup>3</sup> benzene in the air inside

the bus were measured. Smoking was not allowed either in the office building or in the bus. Our findings of benzene in the outdoor air of the city of Munich help to explain why the benzene levels found in blood and exhaled air of the nonsmokers were as high as were observed

2023380496

upon their admission to the laboratory. The values obtained in this experiment are consistent with published outdoor data (BUA-Stoffbericht 1988; Fishbein 1988; Wallace 1989) and do not represent the extreme concentrations that have been found at certain workplaces (WHO 1987; BUA-Stoffbericht 1988; Fishbein 1988; Norpoth 1989). As in indoor environments, the difference between benzene concentrations in buses where smoking was prohibited or allowed was small. In the air of smoking-prohibited buses a median level of  $14 \mu\text{g}/\text{m}^3$  (range  $4\text{--}25 \mu\text{g}/\text{m}^3$ ) was measured, while the value in smoking-allowed buses was  $12.1 \mu\text{g}/\text{m}^3$  (range  $1.4\text{--}49 \mu\text{g}/\text{m}^3$ ) (Proctor et al. 1990 b).

As stated previously, benzene is an established human carcinogen (WHO 1987; IARC 1988; Gesundheitsschädliche Arbeitsstoffe 1989). This has been revealed in several epidemiological studies of workers exposed to high concentrations of benzene over longer periods (Goldstein 1985; WHO 1987; Austin et al. 1988). In animal studies, multipotential carcinogenic effects of benzene were found after high exposure doses ( $> 32 \text{ mg}/\text{m}^3$ ) (WHO 1987). For the most part, they were limited to nonhaematological tissues. In rats and in mice, for example, benzene appears to be strongly related to the occurrence of carcinoma of the zymbal gland, a structure with no human analogue (Maltoni et al. 1985; Austin et al. 1988; Huff et al. 1989).

Whether these findings in man and in animals allow for extrapolation to the general population is questionable (Austin et al. 1988). The human body metabolizes and excretes with no apparent harm low levels of toxic substances that can be quite harmful at higher levels. Humans are well protected against toxicity at low doses from both man-made and natural chemicals. Thus, human exposure to low doses of chemicals that are carcinogenic in rodent bioassays appears to be more common and less hazardous than is generally thought (Ames 1990). As could be shown (Fig. 1), benzene uptake under ETS conditions is neglectable on the background of the body burden caused by other environmental sources. Therefore, it is highly questionable whether it poses a health risk to the general population. In spite of this conclusion, in the effort to improve overall air quality, a further reduction of benzene and related compounds in our environment is desirable.

## References

- Ames BN (1990) Mutagenesis and carcinogenesis: exogenous and endogenous factors. Symposium 13: endogenous versus exogenous factors as major cancer risk determinants. *Am Assoc Cancer Res* 31:512.
- Angerer J, Scherer G, Schaller KH, Müller J (1990) The determination of benzene in human blood as an indicator of environmental exposure to volatile aromatic compounds. *Fresenius Journal of Analytical Chemistry* (in press)
- Aringer L, Lidums V (1988) The influence of diet and other factors on urinary levels of thioethers. *Int Arch Occup Environ Health* 61:123-130.
- Austin H, Deizell E, Cole P (1988) Benzene and leukemia. A review of the literature and a risk assessment. *Am J Epidemiol* 127:419-439.
- Becher H, Jahn I, Jöckel K-H, Bödeker W (1987) Passivrauchen als Gesundheitsrisiko (Ministerium für Arbeit, Gesundheit und Soziales des Landes NRW). Busse Druck, Herford.
- BUA-Stoffbericht 24: Benzol (1988) Hrsg.: Bayer E. Vorsitzender des Beraterpremises für Umweltrelevante Altstoffe (BUA) der Gesellschaft Deutscher Chemiker 24. VCH, Weinheim. pp 64-66.
- Eudy LW, Thomas FA, Heavner DL, Green CR, Ingebretsen BJ (1986) Studies on the vapor-particulate phase distribution of environmental nicotine by selective trapping and detection methods. *Proceedings of the Air Pollution Control Association 79th annual meeting*, pp 2-14.
- Fishbein L (1988) Benzene: uses, occurrence and exposure. In: Fishbein L, O'Neill IK (eds) Environmental carcinogens. Methods of analysis and exposure measurement: vol 10. Benzene and alkylated benzenes. IARC Scientific Publications no. 85. IARC, Lyon. pp 67-96.
- Gesundheitsschädliche Arbeitsstoffe: Toxikologisch-arbeitsmedizinische Begründung von MAK-Werten. In: Heuschler D (ed) Benzol. VCH, Weinheim. 1989. pp 1-53.
- Goldstein BD (1985) Risk assessment and risk management of benzene by the environmental protection agency. In: Hoel DG, Merrill RA, Peters FP (eds) Risk quantitation and regulatory policy. Banbury Report. Cold Spring Harbor Laboratory.
- Grimmer G, Naujick KW, Detibaris G (1987) Gaschromatographic determination of polycyclic aromatic hydrocarbons, azarenes, aromatic amines in sidestream smoke of cigarettes. *Toxicol Lett* 35:117-124.
- Haley NJ, Sepkovic DW, Brunnemann KD, Hoffmann D (1989) Biomarkers for assessing environmental tobacco smoke uptake. In: Harper JP (ed) Combustion process and the quality of the indoor environment. Air and Waste Management Association, Pittsburgh. pp 201-213.
- Heinoonen T, Kyrtöniemi V, Sorsa MI, Vainio H (1983) Urinary excretion of thiocetones among low-tar and medium-tar cigarette smokers. *Int Arch Occup Environ Health* 52:11-16.
- Hengen N, Hengen M (1978) Gas-liquid chromatographic determination of nicotine and cotinine in plasma. *Clin Chem* 24:50-53.
- Hiller FC, McCusker KT, Mazumder MK, Wilson JD, Bone RC (1982) Deposition of sidestream cigarette smoke in human respiratory tract. *Am Rev Respir Dis* 125:406-408.
- Hinds W, First MW, Huber GL, Shea JW (1983) A method for measuring respiratory deposition of cigarette smoke during smoking. *Am Int Hyg Assoc J* 44:113-118.
- Hoffmann D, Brunnemann KD, Hoffmann J (1989) Significance of benzene in tobacco carcinogenesis. *Adv Mod Environ Toxicol* 16:99-130.
- Huff JE, Haseman JK, DeMarini DM, Eustis S, Maronpot RR, Peters AC, Persing RL, Chirisp CE, Jacobs AC (1989) Multipoint carcinogenicity of benzene in Fischer 344 rats and B6C3F<sub>1</sub> mice. *Environ Health Perspect* 82:125-163.
- International Agency for Research on Cancer (1986) Tobacco smoking. IARC Monographs on the evaluation of the carcinogenic risk of chemicals to humans: vol 38. IARC, Lyon.
- International Agency for Research on Cancer (1988) Fishbein L, O'Neill IK (eds) Environmental carcinogens. Methods of analysis and exposure measurement: vol. 10. Benzene and alkylated benzenes. IARC Scientific Publications no. 85. IARC, Lyon.
- Kiefer JE (1978) Ventilated filters and their effect on smoke composition. *Recent Adv Tobacco Sci* 4:69-111.
- Klus H, Begutter H, Ball M, Iatorp M (1987) Environmental tobacco smoke in real life situations. In: Seifert et al (eds) Indoor Air '87. Proceedings of the 4th International Conference on Indoor Air Quality and Climate: vol 1. Volatile organic compounds, combustion gases, particles and fibres, microbiological agents. Institute for Water, Soil and Air Hygiene, Berlin.
- Krause C, Mailahn W, Nagel R, Schulz C, Seifert B, Ulrich D (1987) Occurrence of volatile organic compounds in the air of 500 homes in the Federal Republic of Germany. In: Seifert et al (eds) Indoor Air '87. Proceedings of the 4th International Conference on Indoor Air Quality and Climate. vol 1. Volatile or-

2023380497

- ganic compounds, combustion gases, particles and fibres, microbiological agents. Institute for Water, Soil and Air Hygiene, Berlin.
- Kuwata K, Uebori M, Yamasaki H, Kuge Y (1983) Determination of aliphatic aldehydes in air by liquid chromatography. *Anal Chem* 55:2013-2016
- Langone JJ, Gjika HB, Van Vunakis H (1973) Nicotine and its metabolites: radioimmunoassay for nicotine and cotinine. *Biochemistry* 12:5025-5030
- Maltoni C, Conti B, Cotu G, Belpoggi F (1985) Experimental studies on benzene carcinogenicity at the Bologna Institute of Oncology: current results and ongoing research. *Am J Indust Med* 7:415-446
- Martin F, Hoepfner I, Scherer G, Adlikofer F, Dettbarn G, Grimmer G (1989) Urinary excretion of hydroxy-phenanthrenes after intake of polycyclic aromatic hydrocarbons. *Environ Int* 15:41-47
- NIOSH Manual of Analytical Methods. Method 1500/1501 (1984) US Department of Health and Human Services, vol 1, pp 84-100
- Norpoth K (1989) Benzol. In: Henschler D, Lehnert G (eds) Biologische Arbeitsstoff-Toleranz-Werte (BAT-Werte). B 1, 1-18
- Ogden MW (1989) Gas chromatographic determination of nicotine in environmental tobacco smoke: collaborative study. *J Assoc Off Anal Chem* 72:1002-1006
- Oldaker III GB, Perfetti PF, Conrad FC Jr, Conner JM (1990) Results from surveys of environmental tobacco smoke in offices and restaurants. In: Kasuga (ed) Indoor air quality. Springer, Berlin Heidelberg New York, pp 99-104
- Pellizzari ED, Zweidinger RA, Sheldon LS (1988) Breath sampling. In: Fishbein L, O'Neill IK (eds) Environmental carcinogens. Methods of analysis and exposure measurement: vol 10. Benzene and alkylated benzenes. IARC Scientific Publications no. 85, IARC, Lyon, pp 255-266
- Proctor CJ, Warren ND, Bevan MAJ (1989) Measurements of environmental tobacco smoke in an air-conditioned office building. *Environ Technol Lett* 10:1003-1018
- Proctor CJ, Warren ND, Bevan MAJ, Baker-Rogers J (1990a) A comparison of methods of assessing exposure to environmental tobacco smoke in non-smoking British women. *Environ Int* (in press)
- Proctor CJ, Warren ND, Bevan MAJ, Baker-Rogers J (1990b) Measurements of airborne substances in single-decker buses with and without smoking prohibition. *Anal Chem* (in press)
- Scherer G, Westphal K, Biber A, Hoepfner I, Adlikofer F (1987) Urinary mutagenicity after controlled exposure to environmental tobacco smoke (ETS). *Toxicol Lett* 35:135-140
- Scherer G, Coaze C, Meyerlaak Lv, Sora M, Adlikofer F (1990) Importance of exposure to gaseous and particulate phase components in tobacco smoke in active and passive smokers. *Int Arch Occup Environ Health* (in press)
- Snyder CA (1987) Benzene. In: Snyder R (ed) Ethel Browning's toxicity and metabolism of industrial solvents, 2nd edn vol 1. Hydrocarbons. Elsevier, Amsterdam
- Sterling TD, Dimich H (1982) Indoor byproduct levels of tobacco smoke: A critical review of the literature. *J Air Pollut Control Assoc* 32:250-259
- Surgeon General, A Report of the U.S. Department of Health and Human Services (1986) The health consequences of involuntary smoking. U.S. Government Printing Office, Washington DC
- Wallace LA (1989) Major sources of benzene exposure. *Environ Health Perspect* 82:163-169
- World Health Organisation (1987) Air quality guidelines for Europe. WHO Regional Publication, European Series no. 23. World Health Organization, pp 45-58

2023380498

## Exposure of Passive Smokers to Tobacco Smoke Constituents\*

C. Hugod<sup>1</sup>, L. H. Hawkins<sup>2</sup>, and P. Astrup<sup>1</sup>

\* Dept. Clinical Chemistry, Rigshospitalet, DK-2100 Copenhagen, Denmark

<sup>2</sup> Dept. Human Biology and Health, University of Surrey, Guildford, Surrey, England

**Summary:** In an unventilated room (with or without the presence of ten volunteers) the atmosphere was polluted with sidestream smoke from cigarettes or with the gasphase or constituents of the gasphase of sidestream smoke. One control experiment with no intended air pollution was performed.

The air concentrations of carbon monoxide, carbon dioxide, nitrogen oxides, cyanide, acrolein, other aldehydes, nicotine, and total particulate matter were measured.

By intermittent addition of freshly generated smoke over the three hour experimental period a constant air concentration of 20 ppm carbon monoxide was sustained. When no persons were present, the air concentration of the other measured tobacco smoke constituents remained constant. When persons were present, however, air concentrations of both gasphase and particulate phase constituents decreased during the experimental period.

A considerable variation in the degree of exposure of the passive smokers to the various tobacco smoke constituents was found.

In some of the experiments questionnaires concerning subjective annoyance, eye-, nose- and throat irritation were completed by the subjects. Stay in a gas-phase polluted atmosphere was found equally annoying as in an atmosphere polluted with whole sidestream smoke. Air pollution with acrolein caused considerably less discomfort and this did not differ from the annoyance caused by staying in the closed, unventilated room with no intended air pollution.

It is pointed out that in spite of an often considerable subjective discomfort, exposing non-smokers to tobacco smoke under realistic conditions will not cause inhalation of such amounts of the components of tobacco smoke traditionally considered harmful, that a lasting, adverse health effect in otherwise healthy, grown-up individuals seems probable.

**Key words:** Passive smoking – Health hazards.

This work was supported by a grant from Forschungsgesellschaft Rauchen und Gesundheit, D-2000 Hamburg, Federal Republic of Germany.

0304-0131/78/0042/0021/\$ 1.80

2023380499

Through recent years the harmful effect of tobacco smoking has been documented in a number of primarily epidemiological investigations. The term "passive smoking", i. e., exposure to tobacco smoke of non-smokers in the environment of "active" smokers, was introduced by Steinfeld [22] and since then there seems to have been an increasing public agreement about the fact that tobacco smoke has an adverse health effect on passive smokers [3, 8].

The purpose of the present study has been to investigate whether exposure to other tobacco smoke constituents than carbon monoxide (CO) is at the same level as CO-exposure, i.e., whether air CO-values are representative when estimating the degree of exposure of the passive smoker to other tobacco smoke constituents. Further, by means of questionnaires we sought a subjective rating of eye-, nose-, and throat irritation and the subjective general annoyance in volunteers, who as passive smokers stayed for three hours in a closed, unventilated room with or without air pollution deriving from tobacco smoke or tobacco smoke constituents.

### Material and Methods

Six experiments each of three hours duration were conducted. Five experiments 1, 2, 4, 5, and 6) with participation of volunteers and one (experiment 3) without. In each of the experiments 1, 2, 4, 5, and 6 ten volunteers took part. Altogether 32 persons have participated, only one person took part in all 5 exposure studies. In each of these studies 2-5 of the experimental persons were smokers, 5-8 were non-smokers. The smokers were instructed to refrain from smoking for at least 18 hours before the start of the experiment. The experimental room was unventilated, the windows sealed with tape; the volume was 68.1 m<sup>3</sup>.

**Experiment 1.** Initially 20 cigarettes were "smoked" by machine for 10 minutes resulting in an air concentration of CO of 20 parts per million (ppm). The CO-concentration was now kept at this level by "smoking" further 30 cigarettes during the experimental period. Subjects were admitted to the room as soon as the CO-concentration had reached 20 ppm. (Time (t) = 0).

The air concentrations of the gas-phase constituents: CO, carbon dioxide (CO<sub>2</sub>), cyanide (HCN) and aldehydes (including acrolein), and constituents of the particulate phase: nicotine and total particulate matter (TPM) were measured at different intervals during the experiment. Air temperature and relative humidity were measured and capillary blood samples (from ear lobe) were drawn for determination of COHb.

Questionnaires were given to the subjects at t = 0 and thereafter every 30 minutes during the experimental period. After each one had been completed it was immediately taken back. Four questions sought a subjective rating, from "none" to "very strong" of nasal irritation, eye irritation, throat irritation and "general annoyance", respectively. The subjects were given verbal instructions at the start of the experiment on the use of these assessments.

**Experiment 2.** Identical to experiment 1, apart from nitrogen oxides (NO, NO<sub>2</sub>), but not CO, being measured in this experiment.

**Experiment 3.** Identical to experiment 2, but without the participation of experimental persons. Altogether 50 cigarettes were "smoked".

**Experiment 4.** Subjects were exposed to the gasphase of tobacco smoke only, since the particulate phase (TPM and nicotine) was electrostatically retained in a high voltage filter mounted on the smoking machine. Otherwise this experiment was identical to experiment 1 without measurement of CO<sub>2</sub>.

**Experiment 5.** Subjects were exposed to acrolein in an air-concentration three times as high as in experiment 1 and 2. A known amount of acrolein was evaporated to the atmosphere after heating to the boiling point and mixed with the atmosphere of the experimental room by means of a ventilator.

2023380500

**Table 1.** Analysis of atmosphere in experiment 2 (with people present). Exposure to whole sidestream smoke

Time minutes	CO ppm	NO ppm	NO <sub>x</sub> ppm	Acrolein mg/m <sup>3</sup>	Aldehydes mg/m <sup>3</sup>	HCN mg/m <sup>3</sup>	TPM mg/m <sup>3</sup>	Nicotine mg/m <sup>3</sup>
0	20	0.60	0.00					
30	20	0.34	0.00					
60	22	0.38	0.00	0.048	0.622	0.010		
90	22	0.35	0.00				5.75	0.102
120	22	0.28	0.00					
150	20	0.35	0.00	0.025	0.510	0.014		
180	18	0.30	0.00	0.010	0.391			

**Experiment 6.** Control experiment. Subjects were not exposed to any intended air pollution, but stayed in the room for three hours. Air temperature, relative humidity and CO<sub>2</sub> in air were measured and questionnaires completed.

**Smoking Machine and Cigarettes.** "American type" filter cigarettes were smoked on smoking machine RM 30 (Heinrich Borgwaldt, Hamburg, Germany) without suction (sidestream smoke only). The machine is equipped with a high-voltage "filter" which electrostatically separates the gas-phase and particulate phase of tobacco smoke.

CO was continuously monitored with a "Uta-7n" infrared gas analyzer.

**Nitrogen Oxides.** were continuously monitored with a "Bendix nitrogen oxide analyzer" from which values for NO and NO<sub>x</sub> can be directly obtained.

CO<sub>2</sub> was measured by bubbling an air sample of 10 l (667 ml/min for 15 minutes) through 50 ml 0.15 mol/l Ba(OH)<sub>2</sub> with subsequent titration with 0.1 mol/l HCl.

HCN was measured by bubbling an air sample of 45 l (500 ml/min for 90 minutes) through 100 ml 0.1 mol/l NaOH with subsequent estimation of cyanide by the method described by Elkin [19].

Aldehydes were measured by passing 109 l of air through ice-cooled methanol over a period of 50 minutes. The total volatile aliphatic aldehydes were then estimated colorimetrically by the method of Harke et al. [12], as described by Rothwell and Grant [19].

Acrolein was measured by passing an air sample of 30 l (500 ml/min for 60 minutes) through 4-(n-hexyl)-resorcin with subsequent colorimetric estimation [12].

Nicotine was estimated after passing a total of 2540 l of air through a Cambridge filter over the three-hour period. Nicotine was extracted and determined by gas chromatography.

TPM was determined gravimetrically on the same filter. The concentrations of nicotine and TPM in relation to time can therefore not be estimated.

COHb was measured on a capillary blood sample from the ear lobe by a spectrophotometric method based on Buchwald's [5] modification of the method of Commins and Lawther [7].

## Results

**Experiment 1 and 2.** The results of the measurements in the two experiments were very much alike, and therefore only the results from experiment 2 are shown (Table 1). It can be seen that with people in the room the concentrations of all constituents of the gas-phase (except for HCN) decreased in spite of the fact that freshly generated smoke was added to the atmosphere throughout the exposure period in order to keep CO at a constant level. During the experiment room temperature increased by 3.5°C, air humidity from 56 to 83 relative % and CO<sub>2</sub> concentration to approximately 0.18% (normal 0.03%).

COHb at the start of exposure was 0.73 ± 0.16% (SD, n = 10) (experiment 1 and 2). After 1 hour: 1.16 ± 0.35% (SD), after 2 hours: 1.40 ± 0.26% (SD), and

2023380501

**Table 2. Analysis of atmosphere in experiment 3 (without people present). Whole sidestream smoke**

Time minutes	CO ppm	NO ppm	NO <sub>x</sub> ppm	Acrolein mg/m <sup>3</sup>	Aldehydes mg/m <sup>3</sup>	HCN mg/m <sup>3</sup>	TPM mg/m <sup>3</sup>	Nicotine mg/m <sup>3</sup>
0	21	0.48	0.0					
30	23	0.50	0.0	0.133	1.37	0.050		
60	25	0.59	0.0					
90	25	0.50	0.0		1.10		7.63	0.13
120	24	0.54	0.0					
150	21	0.48	0.0	0.119	failed	0.050		
180	21	0.48	0.0					

**Table 3. Analysis of atmosphere in experiment 4 (with people present). Exposure to gaseous phase only**

Time minutes	CO ppm	NO ppm	NO <sub>x</sub> ppm	Acrolein mg/m <sup>3</sup>	Aldehydes mg/m <sup>3</sup>	HCN mg/m <sup>3</sup>	TPM mg/m <sup>3</sup>	Nicotine mg/m <sup>3</sup>
0	23	0.40	0.02		1.35			
30	23	0.38	0.02	0.19		0.086		
60	24	0.31	0.02					
90	25	0.35	0.02		1.29		Traces	Traces
120	26	0.34	0.01		1.31		0.082	
150	26	0.34	0.03	0.13		1.32		

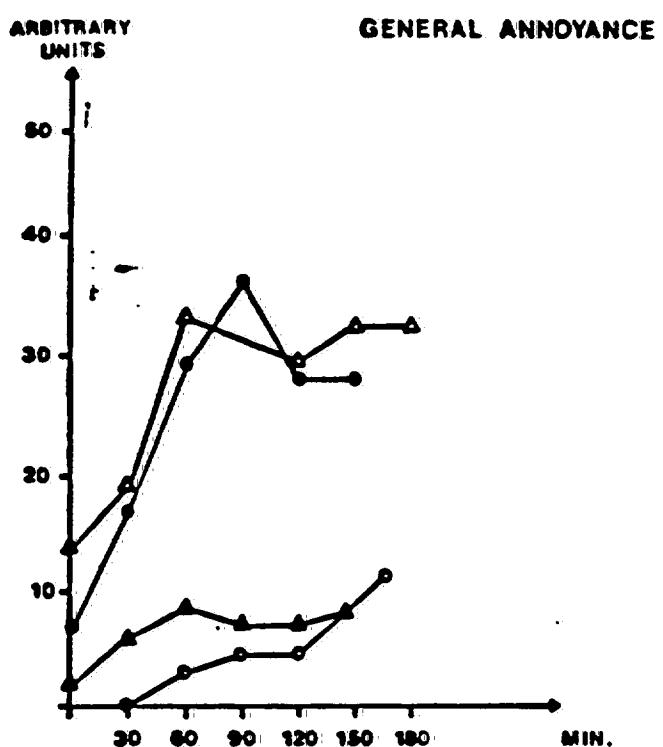
after 3 hours:  $1.63 \pm 0.25$  % (SD). The average increase during the experiment thus was 0.9 % (range 0.5–1.4 %).

**Experiment 3:** From Table 2 it can be seen that the concentrations of all gasphase constituents remain almost constant through the experimental period, without people present. Further, the concentrations of all gasphase constituents are higher without than with people (Tables 1 and 2). Similarly, the measured amounts of TPM and nicotine are higher when people are absent. This suggests that these substances are being removed by the people in the room although it remains uncertain whether this represents a respiratory uptake or a condensation of the components onto skin, hair, and clothing.

**Experiment 4:** When the gasphase is separated from the particulate phase a slight modification in constitution of the gasphase takes place (Table 3). Small amounts of NO<sub>x</sub> are now detected and further – in spite of the presence of people – acrolein, aldehydes and HCN appear in slightly higher concentrations than those found in experiment 3 (sidestream smoke without people present). This suggests that these components under normal conditions are fixed to smoke particles and therefore avoid determination by the applied chemical methods.

**Experiments 5 and 6.** These experiments were performed in order to try to evaluate the reasons for the subjective discomfort experienced by the subjects in the previous experiments, and completing the questionnaires was a way of quantitating this discomfort. 'General annoyance' and eye irritation were at almost identical levels and these were considerably higher than the values for nose- and throat irritation. From

2023380502



**Fig. 1. General annoyance of passive smokers in experiment 2 ( $\Delta-\Delta$ ), 4 ( $\circ-\circ$ ), 5 ( $\bullet-\bullet$ ), and 6 ( $\blacksquare-\blacksquare$ )**

Fig. 1 (for "general annoyance") it can be seen that a maximum value is reached in approximately one hour, and the irritation then remains at this level for the remaining part of the experiment. It is noteworthy that 'general annoyance' as experienced by exposure to the gasphase of tobacco smoke is at the same level as that derived from whole sidestream smoke, and further that exposure to acrolein (in concentrations three times as high as those in experiment 1 and 2) causes only slight discomfort. The curves demonstrated in Fig. 1 were almost identical to those obtained for eye irritation.

#### Discussion

It is often difficult to compare results such as those reported in this study with the findings of others, since different brands of cigarettes smoked in different types of rooms with varying ventilation will lead to different air levels of smoke constituents. These levels will depend first of all on the numbers of cigarettes smoked but also on how the smoking is performed.

2023386503

The smoke available to the passive smoker is a variable mixture of sidestream and exhaled mainstream smoke.

Hoegg [14] has demonstrated that for most of the important constituents sidestream smoke yields about 3–4 times the quantity found in mainstream smoke. HCN seems to be excepted from this rule as the concentration in sidestream smoke is only half of that found in mainstream smoke [4]. This means, that since we in the present study (experiments 1, 2, 3 and 4) created sidestream smoke only from 50 cigarettes, the situation – except for HCN – was worse than it would have been if the cigarettes had been smoked by cigarette-smokers.

In order to increase the degree of exposure we purposely – as much as possible – sought to reduce room ventilation during the experiments. It is possible to calculate the air replacement in the room from the measured rise in CO<sub>2</sub> as compared to what might be expected from the CO<sub>2</sub> production from 10 individuals and the burning cigarettes. This indicates that in experiment 1 the air exchange rate was 41, 22 and 55 % in the first, second and third hour; the differences being attributable to more frequent opening of the door in the first and especially the last hours. These figures are confirmed by similar calculations for CO<sub>2</sub> loss from the room which for experiments 1, 2 and 3 give an average value of 43 %.

The COHb level at the beginning of our experiment was 0.73 %, which might be expected from endogenous CO production. This rose to 1.63 % during the experiment, which is the rise one might expect from smoking and inhaling one cigarette. Because of the experimental situation the rise is probably greater than it would have been after three hours passive smoking under usual conditions. Residing in a large city may cause a similar rise in COHb [6, 11, 16, 23]. The measured levels of other tobacco smoke constituents agree with those of other authors, correcting for differences in room size, ventilation, type of smoke (mainstream/sidestream) and the presence or absence of people [12, 15, 25]. As far as we are aware, HCN concentrations have not been previously measured in a passive smoking experiment. In accordance with Jermini et al. [15] NO was the only nitrogen oxide that could be detected in pure sidestream smoke.

The concentrations of tobacco smoke constituents in air depended on the presence or absence of people in the room. When people were present the average concentrations of NO, aldehydes, acrolein and HCN were reduced by approximately 25, 50, 75, and 80 %, respectively. Further the concentration of acrolein and aldehydes showed a tendency to decline during the experimental period when people were present.

Changes in air-concentrations of TPM and nicotine during the experiment could not be demonstrated since these compounds were determined on samples continuously collected during the whole experimental period. However, the collected amounts of these smoke-constituents were reduced by about 25 % in the experiments with as compared to without persons. A physiological respiratory uptake may explain this dissipation of TPM and nicotine, since the respiratory volume of 10 persons at rest for three hours amounts to about 18–20 % of the volume of the room. The accelerated dissipation of acrolein and aldehydes during the experiment is only partly explained by respiratory uptake and partly by condensation of these components (together with HCN) onto hair, skin, and clothing and fixation to smoke particles.

2023380504

## Passive Smoking

**Table 4.** Mean CO yield from a main stream smoke is taken from Hoegg [14]. Nicotine and TPM yield is taken from analytical data from cigarettes used in this study. The average yields of other compounds are taken from Health Consequences of Smoking 1972 (p. 144-145) [24]. Inhaled amount (mg/h) =  $0.4 \times a$ , where  $0.4 \text{ m}^3/\text{h}$  is taken to be the average respiratory volume of a man at rest,  $a$  is the time weighted mean concentration of each air constituent from experiment 2 (mg/m<sup>3</sup>). CE/h =  $\frac{0.4 \times a}{b}$ , where  $b$  is the average yield of mainstream smoke in mg per cigarette (column 1).

	Average yield from mainstream smoke inhaled by a smoker mg/cig.	Inhaled amount in experiment 2 mg/h	CE/h	CET <sup>a</sup> h
NO	0.30	0.182	0.61	1.6
CO	18.40	9.16	0.50	2.0
Aldehyde	0.81	0.214	0.26	3.8
Acrolein	0.09	0.013	0.14	7.1
TPM	25.30	2.300	0.09	11.1
Nicotine	2.10	0.041	0.02	50.0
Cyanide	0.25	0.005	0.02	50.0

<sup>a</sup> CET = cigarette equivalent time, see text

Other authors have shown [12, 14] that TPM, nicotine, acrolein and aldehydes dissipate even when people are absent. Nicotine dissipates most rapidly and the presence of people accelerated this loss [12].

The concentrations of smoke constituents measured in this study were far below recommended exposure limit values. Relatively highest were acrolein and nicotine (experiment 2) at 20% of the limit value.

Calculations of cigarette equivalents per hour (CE/h) is a conventional way of expressing the amount of a particular component of tobacco smoke inhaled by passive smokers, relative to the amount of the same component inhaled by the active smokers with mainstream smoke from one cigarette.

CE/h can be calculated for each component of the smoke, but because of the various dissipation rates for the different smoke constituents, not for whole tobacco smoke and neither for the whole gasphase of the smoke. Table 4 gives the CE/h values for the constituents measured in experiment 2. Cigarette-equivalent-time (CET, Table 4) expresses the time in hours for which a person has to stay in a room with an atmospheric constitution as in experiment 2, to inhale the same amount of each tobacco smoke constituent, as the "average" cigarette smoker inhales with mainstream smoke from one cigarette.

It appears from Table 4 that a person must stay in the room for two hours to inhale the same amount of CO as is inhaled by active smoking of one cigarette; the corresponding value for HCN is 50 hours. The great differences within the CET values (and CE·h values) are noteworthy and illustrates that it is impossible directly to use the air concentration of one constituent to estimate the degree of exposure to others. Comparison between the CE/h values calculated in this study and corresponding values reported in literature [13, 20] cannot be done directly, because the reported air concentrations differ from those measured in this study.

2023380505

Russell et al. [20] working with twice our exposure level for CO reported a CET-value for CO of 1.0, while ours is 0.5. This difference could be expected because of the different air concentrations of CO.

The present study confirms that many people experience transitory discomfort (local irritation of eye, nose and throat and general annoyance) when staying in an atmosphere heavily polluted with tobacco smoke [3, 8] and it is confirmed that the eye is most sensitive to the irritating effect of tobacco smoke followed by the nose and throat [25]. It is similarly confirmed that staying in an atmosphere polluted with tobacco smoke causes considerable discomfort as expressed by "general annoyance" (Fig. 1).

Further, the results of the subjective assessments suggest that the irritating effect of smoke is confined to constituents of the gasphase.

In contrast to the opinion that acrolein is the most important irritating agent in tobacco smoke [25] exposure to acrolein appears to be only slightly irritating. Even as it is indisputable that passive smoking is connected with immediate discomfort that may be accentuated considerably by the concomitant presence of bronchitis, sinusitis, conjunctivitis, etc. It is difficult to document or to render probable that passive smoking under usual conditions should have a lasting adverse health effect on otherwise healthy individuals.

An exception from this rule seems to be children of smoking parents [17]. An explanation could be the babies' relatively greater alveolar ventilation. Correspondingly there is no reason to doubt the adverse health effect on babies born to mothers who have been smoking during pregnancy [10, 18]. Allergic phenomena might be an explanation for severe discomfort of exposure to tobacco smoke but this is most probably not a frequent cause for non-smokers to feel impaired in their well-being if exposed to tobacco smoke [21, 24].

An adverse health effect of passive smoking in grownup individuals would not be expected if only regarding the inhaled amounts of those tobacco smoke constituents that through the years have been considered pathogenic: nicotine, HCN, TPM and CO. The CET values for nicotine and HCN are negligible and can be disregarded. For CO and nitrogen oxides there is no reason to assume that these components are inhaled by passive smokers in such amounts that they should be considered pathogenic. However, a rise in COHb to approximately 3 %, which is most unlikely obtained from passive smoking, will decrease the threshold for intermittent claudication and angina pectoris [1, 2] in patients with obliterating arterial disease.

The CET-value for TPM is so high (about 11) that the passive smokers will never inhale more than what equals 1/2-1 cigarette per day.

In our opinion and apart from the exceptions mentioned, no data exist to document that any of the gasphase or particulate phase constituents of tobacco smoke reviewed in this study have a lasting adverse health effect in otherwise healthy individuals subjected to passive smoking.

#### References

1. Anderson, E. W., Andelman, R. J., Strauch, J. M., Portier, N. J., Knelson, J. H.: Effect of Low-level Carbon Monoxide on Onset and Duration of Angina Pectoris. Ann. Intern. Med. 79: 46-50 (1973).

2023380506

2. Aronow, W. S., Stenmer, E. A., Isbell, M. W.: Effect of Carbon Monoxide Exposure on Intermittent Claudication. *Circulation* 49, 415-417 (1974).
3. Al ryge eller ikke ryge. Hjerteresningen. Oplysnings-pjæce nr. 11 København (1976).
4. Brunnemann, K. D., Yu, L., Hoffmann, D.: Chromatographic Determination of Hydrogen Cyanide and Cyanogen in Tobacco smoke. *World Smok. Hlth.* 2, 23-28 (1977).
5. Buchwald, H.: A rapid and sensitive Method for Estimating Carbon Monoxide in Blood and its Application in Problem Areas. *Am. Ind. Hyg. Assoc. J.* 30, 564-569 (1969).
6. Cashden, C. M., Cole, P. V.: Variations in Carboxyhaemoglobinlevels in smokers. *Br. med. J.* IV, 736-738 (1974).
7. Commins, B. T., Lowther, P. J.: A Sensitive Method for the Determination of Carboxyhaemoglobin in a Finger Prick sample of Blood. *Br. J. Ind. Med.* 22, 139 (1965).
8. Egsmose, T., Heuer, A.: Handbog for ikke-rygere. Fadl's forlag, København (1977).
9. Elkins, H. B.: The Chemistry of Industrial Toxicology. 2. edition, p. 345. New York: Wiley, 1959.
10. Feichter, L. D., Annau, Z.: Toxicity of Mild Prenatal Carbon Monoxide Exposure. *Science* 197, 680-683 (1977).
11. Göthe, C.-J., Fristedt, B., Sundell, L., Kolmodin, B., Ehrner-Samuel, H., Göthe, K.: Carbon Monoxide Hazard in City Traffic. *Arch. Environm. Health* 19, 310-314 (1969).
12. Harke, H. P., Baars, A., Frahm, B., Peters, H., Schultz, Ch.: Zum Problem des Passivrauchens. *Int. Arch. Arbeitsmed.* 29, 323-339 (1972).
13. Hinds, W. C., Fuss, M. W.: Concentrations of Nicotine and Tobacco Smoke in Public Places. *New Engl. J. Med.* 292, 844-845 (1975).
14. Hoegg, U. R.: Cigarette Smoke in Closed Spaces. *Environ. Health Perspect.* 117-128 (Oct. 1972).
15. Jermini, C., Weber, A., Grandjean, E.: Quantitative Bestimmung verschiedener Gasphasenkomponenten des Nebenstromrauches von Zigaretten in der Raumluft als Beitrag zum Problem des Passivrauchens. *Int. Arch. Occup. Environm. Health* 36, 169-181 (1976).
16. Jones, R. D., Commins, B. T., Cernik, A. A.: Blood Lead and Carboxyhaemoglobin Levels in London Taxi Drivers. *Lancet* II, 302-303 (1972).
17. Leading article. *Br. Med. J.* I, 1179-1180 (1977).
18. Longo, L. D.: Carbon Monoxide: Effects on Oxygenation of the Fetus in Utero. *Science* 194, 523-525 (1976).
19. Rothwell, M., Grant, C. C.: Standard Methods for the Analysis of Tobacco Smoke. Research Paper 11, 2. edition. Tobacco Research Council, London 1974.
20. Russell, M. A. H., Cole, P. V., Brown, E.: Absorption by Non-smokers of Carbon Monoxide from Room Air polluted by Tobacco Smoke. *Lancet* I, 576-579 (1973).
21. Savel, H.: Clinical Hypersensitivity to Cigarette Smoke. *Arch. Environ. Health* 21, 146-148 (1970).
22. Steinfeld, J. L.: A Ban on Public Smoking. *Newsweek* 25, 90 (1971).
23. Stewart, R. D., Baratta, E. D., Platte, L. R., Stewart, E. B., Kalbfleisch, J. D., Yierlao, B. V., Rimm, A. A.: Carboxyhemoglobin Concentrations in Blood from Donors in Chicago, Milwaukee, New York and Los Angeles. *Science* 182, 1362-1364 (1973).
24. The Health Consequences of Smoking. A Report to the surgeon General. U.S. Department of Health, Education and Welfare, 1723-0051, 1972.
25. Weber, A., Jermini, C., Grandjean, E.: Irritating Effects on Man of Air Pollution Due to Cigarette Smoke. *Am. J. Public. Health* 66, 672-676 (1976).

Received June 1, 1978 / Accepted June 16, 1978

2023380507

## MEASUREMENT OF ENVIRONMENTAL TOBACCO SMOKE IN AN AIR-CONDITIONED OFFICE BUILDING

C.J. Proctor, N.D. Warren and M.A.J. Bevan

BAT (UK&E) Ltd., Research and Development Centre,  
Regent's Park Road, Southampton, SO9 1PE, UK

### INTRODUCTION

Environmental Tobacco Smoke, ETS, is the complex mixture of chemicals found in air as a specific result of smoking<sup>1</sup>. Some reports have claimed that exposure to ETS is harmful to the health of the non-smoker<sup>2,3</sup>. This issue has been discussed by scientists and doctors for over a decade and, although knowledge has increased over this period, it is still the subject of scientific controversy<sup>4,5</sup>. The claims have primarily been based on combining the results of epidemiological studies that have all been stated to be, when taken individually, inadequate<sup>2,3,5</sup>. Several experts in the field of low-risk epidemiology have stated that it is not possible to draw firm conclusions as to whether or not ETS is harmful to the health of the non-smoker<sup>4,5,6</sup>.

In spite of the continuing debate, there have been calls for the introduction of further restrictions on where smoking can take place<sup>7,8</sup>. Much attention has recently focused on the work place, and in particular on the modern office environment. This paper presents the results from an investigation of the air in offices in a modern air-conditioned office building located in Southern England.

### SAMPLING SITES

The building selected for this study was a 1970s-built office block comprising of around 9,300 square metres of floor space on 16 floors and holding around 350 people. Air conditioning was nominally the same in all areas, with two operating systems (one at the perimeter and one through the core) being controlled through temperature and humidity sensors.

Ten offices were selected to represent the variety of environments within this building. These included open plan space; single and multiple occupier offices with different populations and numbers of smokers. Samples were acquired between 0900 and 1600 hours, each site being visited on five separate occasions. For a particular office, each visit (which lasted one hour) was performed at a different time of the day and on different days in order to avoid any bias from possible temporal variations. Each sample was taken as near as possible to the centre of the office and at approximately head height of a seated person.

### ANALYTICAL CONSIDERATIONS

The following analytes were determined for each visit by methods that have been previously fully described in the literature:

- (a) Nicotine: Collected by drawing air at a rate of 1 litre per minute through a sorbent sampling tube containing XAD-4 resin. Nicotine was subsequently extracted using ethyl acetate (modified with triethylamine) and determined by capillary gas chromatography with nitrogen-phosphorous detection<sup>9</sup>.

2023380508

**Table 1 : Summary data for offices with at least one smoker compared to offices with no smokers**

(Data in  $\mu\text{g}/\text{m}^3$ , apart from CO and  $\text{CO}_2$  which are given in ppm)

	Smokers' Offices		Non-smokers' Offices	
	Arithmetic Mean	Median	Arithmetic Mean	Median
Nicotine	6	3.1	0.6	0.6
Respirable suspended particulates (RSP)	103	91	90	71
UV-RSP	23	24	8	8.8
Carbon monoxide	1.4	1.1	1.2	1
Carbon dioxide	590	600	533	500
Benzene	13	8	12	10
Chlorobenzene	0.4	0.3	0.4	0.4
n-Decane	8	5	6	5
o-Dichlorobenzene	0.4	0.3	0.6	0.6
1,2-Dichloroethane	13	10	14	14
Dodecane	2	2	2	1
Ethyl benzene	12	5	5	4
Limonene	7	5	3	3
n-Octane	47	3	4	2
$\alpha$ -Pinene	4	3	4	4
Styrene	14	7	17	12
Tetrachloroethylene	4	2	4	1
Trichloroethylene	4	2	4	1
Toluene	39	23	25	20
Undecane	5	4	5	5
2-Vinyl pyridine	3	2	1	1
o-Xylene	14	8	12	11
m/p-Xylene	73	49	69	66

An analysis of variance was used to test the differences between smoking and non-smoking areas. Of the volatile chemicals investigated, only ethyl benzene, limonene and n-octane were significantly different at the 95% confidence limit.

202338C509

- (b) **Respirable suspended particulates (RSP):** Air was drawn at 2 litres per minute through a fluoropore membrane filter via an impactor which only allowed particles of less than  $3.5\mu$  to pass. Measurement of RSP was then gravimetric<sup>1</sup>.
- (c) **Ultra-violet respirable suspended particles (UV-RSP):** After weighing, each pad was extracted with methanol and the resulting solution analysed for its adsorbance at 325nm. Calibration with a surrogate standard, 1,1,2,2-tetrahydroxybenzophenone, allows this measure to be an assessment (albeit an overestimate) of the ETS contribution to RSP<sup>10</sup>.
- (d) **Carbon monoxide:** A constant flow sampling pump delivered air to an electrochemical detector. Output from the detector was fed directly to a data logger<sup>2</sup>.
- (e) **Carbon dioxide:** By Dräger tube (CO<sub>2</sub> 0.01%/a; CH30 801) taken 5 minutes prior to the end of each visit.
- (f) **Volatile organic compounds:** Adsorption on Tenax TA by drawing air through a trap at a rate of 10cm<sup>3</sup> per minute. Subsequent thermal desorption, capillary gas chromatography, mass spectrometry. Peaks were both identified (by retention time and mass spectra) and quantified (using the ion count of the base peak of the mass spectrum of each compound) by the mass spectrometer<sup>1</sup>.

## RESULTS

The results comparing the summary data for offices with at least one smoker to offices with no smokers are given in Table 1. All data points have been included and it can be seen that arithmetic means are generally of a higher value than medians due to a skewed distribution of the values.

Comparing median values it is seen, as might be expected, that the air in smokers' offices contains significantly higher levels of nicotine (3.1 as opposed to 0.6 $\mu$ g/m<sup>3</sup> in non-smokers' offices) and UV-RSP (24 as compared to 9 $\mu$ g/m<sup>3</sup>). RSP values are, on average, higher in smokers' offices, but only by around 20%. The median differences in RSP values between smokers' and non-smokers' offices of 20 $\mu$ g/m<sup>3</sup> is supported by the difference in the UV-RSP values. Hence, ETS is a minor contributor to the respirable particulates found in the air of this building.

There is also little difference in the carbon monoxide and carbon dioxide levels found in smokers' and non-smokers' offices. The median CO value for smokers' offices is 1.1 ppm as compared to 1.0 ppm for non-smokers' offices.

There is little difference between the two categories in the values obtained for the volatile chemicals. For example, benzene in non-smokers' offices had a median value of 10 $\mu$ g/m<sup>3</sup> (arithmetic mean of 12 $\mu$ g/m<sup>3</sup>) whilst in smokers' offices the median value for benzene was 8 $\mu$ g/m<sup>3</sup> (arithmetic mean of 13 $\mu$ g/m<sup>3</sup>). If anything, median values for VOCs tend to be slightly lower in smokers' offices, though arithmetic means tend to be slightly higher.

2023380510

### CONCLUSIONS

This investigation of the chemicals in the air of smokers' and non-smokers' offices in an air-conditioned building results in several conclusions:

1. The concentrations of constituents related to ETS (such as nicotine) were extremely small, even in smokers' offices. On average a non-smoker would have to work in a smoker's office for three months (7 hours per day, 5 days per week) to be exposed to the equivalent nicotine from one cigarette.
2. By comparing smokers' and non-smokers' offices and by observing values of RSP and UV-RSP (the ETS contribution to particulates) it seems that, in this environment, ETS was a minor contributor to the concentration of respirable particulates in air.
3. The presence of ETS had little influence on the carbon monoxide level in an office.
4. Environmental Tobacco Smoke did not significantly contribute to the concentrations of volatile organic compounds, such as benzene, in the office building investigated.

### KEY REFERENCES

1. C.J. Proctor, Environ. Tech. Lett., **9**, 553-562, 1988.
2. US NRC, ETS: Measuring exposures and assessing health risks, National Academy Press, Washington DC, 1986.
3. Report of the US Surgeon General, US Gov. Printing Office, Washington DC, 1986.
4. N. Mantel, British Med. J., **294** (6569), 440-441, 1987.
5. K. Überla, Int. Arch. Occup. Environ. Health, **9** (5), 21-37, 1987.
6. P.N. Lee, Misclassification of smoking habits and passive smoking, Springer-Verlag, 1988.
7. The Fourth Report of the Independent Scientific Committee on Smoking and Health, Her Majesty's Stationery Office, London, 1988.
8. T.D. Sterling and B. Mueller, Am. Ind. Hyg. Assoc. J., **49** (9), 423-426, 1988.
9. G.B. Oldaker and F.C. Conrad, Environ. Sci. and Technol., **21** (10), 994-999, 1987.
10. M.W. Ogden, 101st Annual Int. Meeting of the Association of Official Analytical Chemists, Sept. 14-17, San Francisco, 1987.

2023380511

illy Deposited  
tion of Occup-  
ed States."  
b Interoffice  
22.  
7). "Ionizing

## Benzene: Environmental Partitioning and Human Exposure<sup>1</sup>

HOLLY A. HATTEMER-FREY,\* CURTIS C. TRAVIS,<sup>†</sup> AND MIRIAM L. LAND<sup>†</sup>

\*Lee Wan and Associates, Inc., 120 S. Jefferson Circle, Suite 100, Oak Ridge, Tennessee 37830;  
and <sup>†</sup>Office of Risk Analysis, Health and Safety Research Division, Oak Ridge National  
Laboratory, P.O. Box 2008, Building 4300S, Oak Ridge, Tennessee 37831-6109

Received April 12, 1990

A multimedia transport model was used to evaluate the environmental partitioning of benzene. Measured and predicted environmental concentrations were used to estimate the accumulation of benzene in the food chain and the subsequent extent of human exposure from inhalation and ingestion. Results show that benzene partitions mainly into air (99.9%) and that inhalation is the dominant pathway of human exposure, accounting for more than 99% of the total daily intake of benzene. Ingestion of contaminated food items represents only a minor pathway of human exposure. The long-term average daily intake of benzene by the general population of the U.S. was estimated using three independent methods. Intake estimates based on measured personal air exposures, measured exhaled air concentrations, and a pharmacokinetically derived adipose tissue concentration (73, 63, and 72 µg/day, respectively) are in good agreement. Although inhalation is the primary route of human exposure to background levels of benzene, the contribution of ingestion to the total daily intake is the largest anthropogenic source of background human exposure to benzene.

### INTRODUCTION

Benzene has the largest production volume of any chemical that has been causally linked to cancer in humans (U.S. EPA, 1984). Benzene is produced commercially as an intermediate in the production of many chemicals and is a by-product of various combustion processes, such as forest fires and the burning of wood, garbage, organic wastes, and cigarettes (Fishbein, 1984; IARC, 1982; Webster *et al.*, 1986). The fact that benzene has been measured in air, water, and human biological samples (Antonie *et al.*, 1986; U.S. EPA, 1986a; Wakeham *et al.*, 1986; Wallace, 1986; Wallace *et al.*, 1987) suggests that environmental contamination of benzene is widespread. This paper evaluates the environmental partitioning of benzene and identifies the major sources of human exposure.

### ENVIRONMENTAL PARTITIONING

Various measurement and predictive techniques can be used to evaluate the movement and transfer of chemicals within and between environmental media. Multimedia partitioning models, for example, estimate the long-term steady-state concentration of pollutants in various media. One such model, the Level III Fugacity model of Mackay *et al.* (1985a, b), treats the environment as a "unit

<sup>1</sup> Research was sponsored by the U.S. Environmental Protection Agency and the American Petroleum Institute under Interagency Agreements applicable under Martin Marietta Energy Systems, Inc., Contract No. DE-AC05-84OR21400 with the U.S. Department of Energy.

2023380512

"world" (a hypothetical region equal to 1 km<sup>2</sup>) divided into six homogeneous compartments: air, water, soil, bottom sediment, suspended sediment (in water), and aquatic biota (fish). Under conditions of continuous release, this model can be used to estimate unknown or nondetectable concentrations in certain media from a chemical's physicochemical properties and from known (detectable) concentrations in other media, thus providing a coherent account of concentrations in all media.

The Fugacity model was modified to account for the uptake of chemicals through the food chain (Hattemer-Frey and Travis, 1989; Travis and Hattemer-Frey, 1987). The modified version, called the Fugacity Food Chain (FFC) model, estimates the concentration of a chemical in all six media and then uses those concentrations to predict the amount of the chemical entering the food chain and the average daily intake by the general population. It should be emphasized, however, that the FFC model is not an exact replica of the environment, since it contains many simplifying assumptions. Given the current state of knowledge concerning the environmental transport of organic chemicals, however, fugacity models are generally considered acceptable for exploring the equilibrium partitioning and environmental behavior of organic chemicals (Allen *et al.*, 1990; Cohen and Ryan, 1985; Mackay *et al.*, 1985a, b).

Input parameters required to predict the cross-medium partitioning of a chemical include (1) its physicochemical properties; (2) estimates of its bioaccumulation potential (i.e., bioconcentration (BCF) and biotransfer (BTF) factors); (3) degradation rates for processes that remove the compound from the system; and (4) an estimate of emissions into air, water, and soil. The physicochemical properties of benzene and its bioconcentration and biotransfer factors are presented in Table 1.

#### Degradation Rate Estimates

Most processes that effectively remove a compound from a medium involve chemical or biochemical reactions (Mackay *et al.*, 1985a). The most significant

TABLE I  
PHYSICOCHEMICAL PROPERTIES OF BENZENE

Physicochemical properties		
Log octanol-water partition coefficient (log $K_{ow}$ )	2.13	Chiou <i>et al.</i> , 1977
Water solubility	22.8 mol/m <sup>3</sup>	Wakeham <i>et al.</i> , 1986
Vapor pressure	12,700 Pa	Mackay <i>et al.</i> , 1985a
Molecular weight	78.11	Mackay <i>et al.</i> , 1985a
Henry's Law constant (H)	$5.5 \times 10^{-3}$	Lyman <i>et al.</i> , 1982
Soil adsorption coefficient	N4 ml/g	Lyman <i>et al.</i> , 1982
Bioconcentration (BCFs) and biotransfer factors (BTFs)		
Air-to-leaf BCF	0.12	Travis & Hattemer-Frey, 1990b
Soil-to-root BCF	2.0	Schuenemeyer <i>et al.</i> , 1985
Daily intake-to-beef BTF	$3.4 \times 10^{-6}$ d/kg	Travis and Arms, 1988
Daily intake-to-milk BTF	$1.1 \times 10^{-6}$ d/kg	Travis and Arms, 1988
Water-to-fish BCF	5.2	US EPA, 1986a

2023380513

homogeneous  
in water),  
model can be  
media from  
(3) concentra-  
rations in all

of chemicals  
d Hattemer-  
FFC model.  
n uses those  
od chain and  
emphasized.  
ent, since it  
f knowledge  
ver, fugacity  
ibrium parti-  
, 1990; Co-

g. of a chem-  
bioaccumula-  
factors); (3)  
system; and  
chemical prop-  
presented in

lum involve  
t significant

-7  
1986  
985a  
985a  
#12  
#2

ier-Frey, 1990b  
1985  
., 1988  
. 1988

degradation pathway of benzene in air is its reaction with atmospheric hydroxy radicals (ATSDR, 1987). U.S. EPA (1986a) reported that benzene photolysis in the atmosphere has a half-life of 6 days. Therefore, a degradation rate coefficient of  $4.8 \times 10^{-3} \text{ hr}^{-1}$  was used to model the removal of benzene from air.

Biodegradation and photolysis are the primary mechanisms of benzene removal from water. Mackay *et al.* (1985a) reported that the biodegradation of benzene in water occurred at the rate of  $4.5 \times 10^{-3} \text{ hr}^{-1}$ , while photolytic degradation of benzene in water took place at the rate of  $1.8 \times 10^{-4} \text{ hr}^{-1}$ . Collectively, these two processes yield a degradation rate coefficient for benzene in water of  $4.76 \times 10^{-3} \text{ hr}^{-1}$ .

The biodegradation of benzene in soil is well-documented (Tucker *et al.*, 1986). A degradation rate coefficient for benzene in soil and sediment of  $2.78 \times 10^{-4} \text{ hr}^{-1}$  (Tucker *et al.*, 1986) was used in this analysis.

#### Emission Rate Estimates

Potential sources of environmental benzene include benzene production, gasoline refining, coal coking, production of benzene-based chemicals, solvent use, oil and chemical spills, leaking storage tanks, and the combustion of gasoline (ATSDR, 1987; U.S. EPA, 1983). Over 60% of emissions from these sources are attributed to the combustion of gasoline (Fishbein, 1984; U.S. EPA, 1983), while 10 to 30% are from stationary industrial sources (Cross *et al.*, 1979; Fishbein, 1984; U.S. EPA, 1984). Annual emissions of benzene in the U.S. alone are estimated to be  $8.5 \times 10^9 \text{ kg}$  (SRI International, 1988).

Although the magnitude of the source term and the exact pattern of environmental release are not known for most pollutants, measured concentrations of a compound in air, water, and soil can be used to estimate the distribution of release, assuming that measured environmental concentrations are in equilibrium with current inputs. Since the network of equations in the FFC model is linear, media concentrations respond proportionally to changes in emission quantities. Thus, measured background concentrations of benzene in air ( $4.6 \mu\text{g}/\text{m}^3$  (Bozzelli and Kebbekus, 1982; Wallace, 1986)) and water ( $10 \text{ ng/liter}$  (Saucer, 1981)) were used to predict the cross-media partitioning of benzene using the methodology described in Travis and Hattemer-Frey (1990b). Since the background concentration of benzene in soil has not been measured, the following equation from Lyman *et al.* (1982) was used to predict this value

$$C_s = (C_w)(K). \quad (1)$$

where  $C_s$  is the estimated concentration of benzene in soil ( $\text{ng}/\text{m}^3$ ),  $C_w$  represents the measured background concentration of benzene in water ( $\text{ng}/\text{m}^3$ ), and  $K$  is a constant that is equivalent to the chemical's soil/water partitioning coefficient ( $K_{oc}$ ) times the total organic carbon content of soil. Using a  $K_{oc}$  value of 84 ml/g for benzene and assuming the total organic carbon content of soil is 10% (Lyman *et al.*, 1982),  $C_s$  is estimated to be  $8.4 \times 10^4 \text{ ng}/\text{m}^3$  or 56 ng/kg.

The FFC model was calibrated by varying emission rate estimates until the concentration of benzene predicted by the model was consistent with measured

2023380514

background concentrations of benzene in air and water and the predicted concentration of benzene in soil. The following emission rates were found to best reproduce the environmental data:

$$\begin{aligned} \text{Air} &= 1.21 \times 10^7 \text{ mol/hr (99.96\%)} \\ \text{Water} &= 4.57 \times 10^3 \text{ mol/hr (0.04\%)} \\ \text{Soil} &= 3.43 \times 10^2 \text{ mol/hr (0.00\%)} \end{aligned}$$

These data show that virtually all (99.96%) of the benzene released into the environment is emitted into the atmosphere. These emissions estimates agree well with the Powell and Tucker (1986) estimate that 95% of environmental releases of benzene are into air. Furthermore, total emissions of benzene into the U.S. environment estimated by the FFC model are  $8.3 \times 10^9 \text{ kg/year}$ , which agrees well with reported production estimates of  $8.5 \times 10^9 \text{ kg/year}$  (SRI International, 1988). Even if only 50% of the benzene produced annually in the U.S. is actually released into the environment, our source term estimate remains within a factor of two of reported production value. This result demonstrates that the FFC model can reliably predict annual emissions using background environmental data.

#### *Environmental Fate of Benzene*

Results show that benzene partitions mainly into the air (99%), with less than 1% partitioning into water, soil, sediment, suspended sediment, and biota. Table 2 gives the predicted environmental concentrations for benzene. These values are approximations that are limited by the accuracy and availability of reported data.

TABLE 2  
COMPARISON OF PREDICTED AND MEASURED ENVIRONMENTAL CONCENTRATIONS FOR BENZENE

Phase	Predicted concentration (ng/kg) <sup>a</sup>	Measured background concentration (ng/kg)	Reference for measured value
Air <sup>b</sup>	4.6	4.8 5.0 4.5	Bozzelli and Kebbekus, 1982 Wallace <i>et al.</i> , 1982 Webster <i>et al.</i> , 1986
Water <sup>b</sup>	10	6.0	Sauer, 1981
Soil	.56	NA	
Sediment	.23	NA	
Suspended sediment	.23	NA	
Fish	.52	NA	
Forage	.587	NA	
Grain	11.2	NA	
Exposed produce	.587	NA	
Root crops	11.2	NA	
Beef	2.5	NA	
Milk	0.8	NA	

<sup>a</sup>  $\mu\text{g/m}^3$ .

<sup>b</sup> ng/liter.

2023380515

Alth  
beer  
dairy p  
used t  
product

Accum  
Acc  
deposit  
benzen  
*et al.*, 198  
air-to-le  
ination  
estimat

where /  
Law co  
(Chiou  
1982), a  
The c  
estimate

where C  
fraction  
tration c  
in the v  
humans

Since  
a major  
vegetati  
tration c  
net et a  
benzene  
value of  
obtained  
Using th  
groups i

Since  
sumed th  
assumed

### ACCUMULATION OF BENZENE IN THE FOOD CHAIN

Although data on the occurrence of benzene in food are scarce, benzene has been reported to occur naturally in many foods including fruits, vegetables, fish, dairy products, beverages, and eggs (Mara & Lee, 1978). The FFC model was used to estimate human exposure to benzene from ingestion of contaminated produce, beef, milk, and fish.

#### *Accumulation on Vegetation*

Accumulation of organics in vegetation is a complex process that can involve deposition, root uptake, and air-to-leaf transfer. Only a negligible fraction of benzene is expected to sorb to particulates in the air (Bidleman, 1988; Mackay *et al.*, 1986) as its primary states are the gaseous and dissolved forms. As a result, air-to-leaf transfer was expected to be the major pathway of vegetative contamination. Although an air-to-leaf BCF has not been measured for benzene, it was estimated using the equation (Travis and Hattomer-Frey, 1990b)

$$B_{va} = 5.0 \times 10^{-6} K_{ow} + H \quad (2)$$

where  $K_{ow}$  is the octanol-water partition coefficient, and  $H$  represents Henry's Law constant in atmospheres per cubic meter per mole. Using a log  $K_{ow}$  of 2.13 (Chiou *et al.*, 1977) and an  $H$  value of  $5.5 \times 10^{-1}$  atm · m<sup>3</sup>/mol (Lyman *et al.*, 1982), a  $B_{va}$  value of 0.12 was used in this analysis.

The concentration in vegetation due to the air-to-leaf transfer (CVA) can be estimated from the equation (Travis and Hattomer-Frey, 1990b).

$$CVA (\text{pg/kg}) = C_a \cdot F_v \cdot B_{va} \quad (3)$$

where  $C_a$  is the concentration of organic in air ( $\mu\text{g}/\text{m}^3$ ), and  $F_v$  represents the fraction of compound that exists in air as a vapor. Using the measured concentration of benzene in air of ( $4.6 \times 10^3 \text{ ng}/\text{m}^3$ ) and assuming that all benzene exists in the vapor phase (i.e.,  $F_v$  equals 1.0), CVA for exposed plants consumed by humans and forage is estimated to be 475 ng/kg.

Since benzene is not very soluble in water, root uptake was not expected to be a major source of vegetative contamination. The concentration of benzene in vegetation due to root uptake (CVR) can be estimated by multiplying the concentration of organic in soil times a chemical-specific soil-to-root BCF ( $B_s$ ). Scheuerert *et al.* (1983) reported that cress and barley grown in soil with a steady-state benzene concentration of 0.005 ppm accumulated 0.01 ppm benzene. Hence, a  $B_s$  value of 2.0 was used in this analysis. This value agrees well with a value of 2.3 obtained using the regression equation developed by Travis and Arms (1988). Using the calculated concentration of benzene in soil (56 ng/kg), CVR for all plant groups is estimated to be 112 ng/kg.

Since benzene is assumed to exist exclusively in the vapor phase, it was assumed that plants were not contaminated by direct deposition. Furthermore, we assumed that protected produce, including potatoes and other root vegetables,

2023380516

legumes, and garden fruits whose edible portions either grow underground or are protected by pods, shells, or nonedible peels, were not contaminated via the air-to-leaf pathway, because there is no empirical data to support the hypothesis that chemical vapors absorbed by aerial plant parts are actually translocated to other plant parts. If air-to-leaf transfer is a surface phenomenon only (i.e., no translocation occurs), then our assumption is valid. If, on the other hand, translocation does occur, then our assumption may not be valid. Given that the food chain is a minor pathway of human exposure to benzene, we do not believe that the assumption of no contamination via the air-to-leaf pathway for protected produce would substantially alter our findings.

The total concentration of benzene in plants is determined by summing the contribution from each of the two pathways of vegetative contamination (CVR + CVA). The total concentration of benzene on exposed food crops consumed by humans and forage is estimated to be 387 ng/kg, 81% of which was due to air-to-leaf transfer and 19% to root uptake. The total concentration of benzene in grain and roots crops is 112 ng/kg. These results demonstrate that air-to-leaf transfer is the primary pathway of vegetative contamination.

#### *Accumulation in Beef, Milk, and Fish*

Since benzene is not very lipophilic ( $\log K_{ow} = 2.13$ ), it was not expected to accumulate to a large extent in living organisms. The concentration of benzene in cow milk and beef can be estimated from Eqs. (6 and 7) in Hattemer-Frey and Travis (1989) using steady-state daily intake-to-cow milk and beef BTFs of  $3.4 \times 10^{-6}$  d/kg and  $1.1 \times 10^{-6}$  d/kg, respectively (Travis and Arms, 1988). The predicted daily intake of benzene by beef and dairy cattle from inhalation and from ingestion of contaminated feed and water is presented in Table 3. These data show that inhalation is the primary pathway of benzene exposure for cattle. The predicted concentration of benzene in beef and dairy products is 2.5 ng/kg and 0.8 ng/kg, respectively (Table 2).

Again, since the bioaccumulation potential of benzene is small, it was not expected to accumulate substantially in fish. For example, the water-to-fish BCF for benzene is only 5.2 (U.S. EPA, 1986a) compared with BCFs of 140,000 for dioxin (Travis and Hattemer-Frey, 1990a) and 60,000 for PCBs (Bruggeman et al.,

TABLE 3  
INTAKE OF BENZENE BY BEEF AND DAIRY CATTLE

	Intake by beef cattle ( $\mu\text{g}/\text{day}$ )	Percentage of total daily intake	Intake by dairy cattle ( $\mu\text{g}/\text{day}$ )	Percentage of total daily intake
Inhalation:	728.0	99.7%	728.0	99.0%
Water	0.02	<0.01%	0.02	<0.01%
Soil	0.004	<0.01%	0.01	<0.01%
Forage	1.36	0.21%	6.45	0.87%
Grains	0.56	0.08%	0.80	0.11%
Total	730.1	100%	735.3	100%

i or are  
via the  
othesis  
ated to  
e., no  
trans-  
e food  
e that  
ected

ng the  
VR -  
ed by  
to air-  
ene in  
o-leaf

ted to  
ene in  
y and  
3.4 x  
e pre-  
from  
show  
: pre-  
d 0.8

< not  
BCF  
0 for  
r al..

ntage  
otal  
ntake

0%  
11%  
11%  
7%  
1%  
41%

1984). Using the measured background concentration of benzene in water (10 ng/kg), the concentration of benzene in fish is estimated to be 52 ng/kg (Table 2).

### QUANTIFYING THE EXTENT OF HUMAN EXPOSURE TO BENZENE

An important objective in risk assessment is elucidating the pathways and extent of human exposure to pollutants chronically released into the environment. Often people are exposed to higher levels of pollutants from sources in the home than from traditional emission sources. The Total Exposure Assessment Methodology (TEAM) studies showed that benzene levels in personal air (the air that humans breathe) averaged two times higher than outdoor air concentrations (Hartwell *et al.*, 1987a, b; Wallace, 1986; Wallace *et al.*, 1982, 1983, 1987). Wallace (1986) found that exposure to benzene concentrations indoors was greater than exposure to benzene levels near gas stations in most cases. Personal air concentrations ranged from 7.3 to 28  $\mu\text{g}/\text{m}^3$ , with a geometric mean concentration of 13.7  $\mu\text{g}/\text{m}^3$ , while outdoor air concentrations ranged from 1.9 to 16  $\mu\text{g}/\text{m}^3$ , with a geometric mean of 5.6  $\mu\text{g}/\text{m}^3$  (Wallace, 1986).

To estimate the average absorbed dose of benzene under steady-state conditions, the mean concentration of benzene in personal air (13.7  $\mu\text{g}/\text{m}^3$ ) (Wallace, 1986) was multiplied by the average adult alveolar ventilation rate (AVR) and the fraction of inhaled benzene that is metabolized under steady-state conditions ( $F_m = 0.38$ ) (Travis *et al.*, 1990b). Assuming that 70% of the total volume of air inspired during light activity (20  $\text{m}^3/\text{day}$ ) is available for gaseous exchange, an AVR of 14  $\text{m}^3/\text{day}$  was used in this analysis (ICRP, 1975). Thus, the average absorbed dose is estimated to be 73  $\mu\text{g}/\text{day}$ . Exposure to maximum personal air concentrations (28  $\mu\text{g}/\text{m}^3$ ) could result in an exposure of 149  $\mu\text{g}/\text{day}$ .

In the current EPA risk assessment process, cancer potency values are based upon administered dose, not absorbed dose. Thus, risk estimates for benzene should be based on the mean concentration of benzene in personal air (13.7  $\mu\text{g}/\text{m}^3$ ) and the average adult inhalation rate (20  $\text{m}^3/\text{day}$ ) (ICRP, 1975). Assuming that the carcinogenic potency of benzene is  $2.6 \times 10^{-3}$  (mg/kg-day)<sup>-1</sup> (U.S. EPA, 1986a), the corresponding excess lifetime cancer risk is  $1 \times 10^{-4}$ .

The amount of benzene taken in from contaminated drinking water was calculated by assuming that the average adult consumes 1.44 liters of water-based beverages daily (Yang and Nelson, 1986). Total dietary intake was calculated by multiplying the estimated concentration of benzene in food items by average adult U.S. consumption rates (Yang and Nelson, 1986). Table 4 gives our estimate of the average daily intake of benzene by the general population of the U.S. These data show that inhalation accounts for more than 99% of human exposure to benzene and that ingestion of contaminated food and water is not a major pathway of human exposure.

The concentration of organic chemicals in human adipose tissue can serve as an indicator of past exposure, since many environmental contaminants bioaccumulate in human tissues (Geyer *et al.*, 1986). To verify our estimate of benzene exposure, a linear, one compartment pharmacokinetic model was used to estimate the long-term, average daily intake ( $I$ ) of benzene according to the equation (Geyer *et al.*, 1986)

2023380518

TABLE 4  
PREDICTED AVERAGE DAILY INTAKE OF BENZENE BY THE GENERAL POPULATION  
OF THE UNITED STATES

Source	Daily intake ( $\mu\text{g}/\text{day}$ )	Percentage of total daily intake
Inhalation	72.90	99.96%
Water	0.01	0.01%
Food (total)	0.02	0.03%
Total	72.93	100%
<b>Food chain intake:</b>		
Produce	0.019	93.1%
Dairy products	0.0002	1.2%
Beef	0.0002	1.1%
Fish	0.001	4.6%
Total	0.02	100%

$$I = \ln 2 (m_{\text{bb}}) / T, \quad (4)$$

where  $m_{\text{bb}}$  is the total body burden, and  $T$  is the half-life of the compound in the human body in days. The half-life of benzene in humans was estimated from a pharmacokinetic model to be approximately 12 hr (0.51 days) (Travis *et al.*, 1990b). Assuming that the average human weighs 70 kg and has 22% body fat (i.e., 15.4 kg of fat) (ICRP, 1975) and 9.9  $\mu\text{g}$  benzene/kg fat (the geometric mean adipose tissue concentration for the general population of the United States as measured by the U.S. EPA (1986b) as part of their National Human Adipose Tissue Survey), the mean total human body burden ( $m_{\text{bb}}$ ) of benzene is estimated to be 153  $\mu\text{g}$ , and the average, long-term daily intake of benzene is estimated to be 207  $\mu\text{g}/\text{day}$ .

There is reason to believe, however, that the 9.9  $\mu\text{g}/\text{kg}$  concentration of benzene in human adipose tissue measured by the U.S. EPA (1986b) may be inaccurate. Using a mean exposure concentration of 13.7  $\mu\text{g}/\text{m}^3$  and an AVR of 14  $\text{m}^3/\text{day}$ , the steady-state concentration of benzene in adipose tissue estimated using a pharmacokinetic model is 3.45  $\mu\text{g}/\text{kg}$  fat (Travis *et al.*, 1990a). This value is nearly a factor of three lower than the concentration measured by the U.S. EPA (1986b). Assuming that the pharmacokinetically based estimate of steady-state human adipose tissue concentrations of benzene is correct, then the mean total body burden of benzene is estimated to be 53  $\mu\text{g}$ , which corresponds to an average, long-term daily intake of 72  $\mu\text{g}/\text{day}$ . This exposure estimate is consistent with the 73  $\mu\text{g}/\text{day}$  intake estimate based on measured benzene levels in personal air (Wallace, 1986).

Exhaled air concentrations can also be used as an indicator of past exposure. The long-term, average daily intake can be calculated using the equation

$$I = C_e \cdot R / (1 - F_m), \quad (5)$$

where  $C_e$  equals the concentration of the compound in exhaled air,  $R$  is the assumed respiration rate (14  $\text{m}^3$  per day), and  $F_m$  is the fraction of the compound

2023380519

## POPULATION

Age of total y intake
0.96%
1.01%
1.03%
100%
13.1%
1.2%
1.1%
4.6%
100%

(4)

compound in the estimated from a (Travis *et al.*, 1990a). 22% body fat geometric mean United States as Human Adipose tissue is estimated estimated to be

concentration of benzene may be inactivated by an AVR of 14 (Travis *et al.*, 1990a). This value is the U.S. EPA estimate of steady-state the mean total tends to an average consistent with in personal air.

past exposure. equation

(5)

air.  $R$  is the the compound

inhaled that is metabolized (0.38) (Travis *et al.*, 1990b). Assuming that mean exhaled air concentrations range from 1.3 to 9.8  $\mu\text{g}/\text{m}^3$ , with a geometric mean concentration of 2.8  $\mu\text{g}/\text{m}^3$  (Wallace, 1986; Wallace *et al.*, 1982, 1985, 1987), the long-term, average daily intake of benzene is estimated to be 63  $\mu\text{g}/\text{day}$  with a range of 29 to 222  $\mu\text{g}/\text{day}$ .

Thus, intake estimates based on measured personal air exposures, measured exhaled air concentrations, and a pharmacokinetically-derived adipose tissue concentration (73, 63, and 72  $\mu\text{g}/\text{day}$ , respectively) are in good agreement. We believe that the estimate based on a human adipose tissue concentration estimated by the U.S. EPA (1986b), 207  $\mu\text{g}/\text{day}$ , is flawed. Nevertheless, all of these estimates are substantially lower than the estimate of 850  $\mu\text{g}/\text{day}$  reported by the National Research Council (1980).

#### Extent of Human Exposure to Benzene from Cigarette Smoking

Smoking is by far the largest anthropogenic source of background human exposure to benzene. Wallace *et al.* (1987) reported that smokers had levels of benzene in exhaled air 2 to 10 times higher than nonsmokers. Travis *et al.* (1990a), using a physiologically based pharmacokinetic model, estimated that the absorbed dose of benzene from inhaling cigarette smoke is 40  $\mu\text{g}$  per cigarette. Their value agrees well with other reported estimates of 30  $\mu\text{g}$  per cigarette (Powell and Tucker, 1986) and 57  $\mu\text{g}$  per cigarette (Higgins *et al.*, 1983; Wallace *et al.*, 1987). Thus, average smokers (i.e., individuals who smoke 20 cigarettes a day) are taking in an additional 800  $\mu\text{g}$  of benzene daily, which means that they get 290% more benzene from smoking than from background environmental contamination. The increased lifetime cancer risk associated with smoking 20 cigarettes per day is  $3 \times 10^{-4}$ . Heavy smokers (i.e., individuals who smoke 35 cigarettes a day) are likely to experience an increased lifetime cancer risk of  $5.2 \times 10^{-4}$  from exposure to benzene (additional intake of 1400  $\mu\text{g}/\text{day}$ ). Thus, average and heavy smokers may experience a total lifetime cancer risk (from background exposure and smoking) of  $3.2 \times 10^{-4}$  and  $5.5 \times 10^{-4}$ , respectively.

Nonsmokers who live with or come in contact with a smoker also have elevated levels of benzene in their breath (Wallace *et al.*, 1987). Nonsmokers who live with a smoker had about 30% to 50% higher benzene levels in their breath than nonsmokers who did not live with a smoker (i.e., mean exhaled air concentrations equal 3.6 to 4.2  $\mu\text{g}/\text{m}^3$ ). The increased lifetime cancer risk for a nonsmoker who lives with a smoker is  $2.8 \times 10^{-5}$  to  $3.3 \times 10^{-5}$ .

#### CONCLUSIONS

Organic chemicals tend to accumulate in the media in which they are most soluble. Our environmental partitioning model showed that benzene, a highly volatile, nonlipophilic compound, partitions mainly into air and does not accumulate to an appreciable extent in the food chain. Quantifying the environmental partitioning and extent of human exposure to benzene is the first application of the FFC model to a highly volatile, nonlipophilic organic (Travis and Hattemer-Frey, 1987; Hattemer-Frey and Travis, 1989), which demonstrates the utility of model-

2023380520

ing efforts to predict the extent of human exposure to environmental contaminants through the food chain.

Ingestion of contaminated food items has been suggested to be a potentially important pathway of human exposure to benzene (NRC, 1980; Gilbert *et al.*, 1982). The NRC (1980) estimated that the average U.S. urban exposure to benzene is 850 µg/day and that the dietary intake of benzene may be as high as 250 µg/day. Gilbert *et al.* (1982) suggested that ingestion of contaminated food items may result in benzene intakes of 31 to 108 µg/day. Our results, however, show that ingestion contributes less than 1 µg/day, which accounts for less than 1% of the average daily intake of benzene by the general population of the U.S. The highest concentration of benzene in food items appears to be in exposed vegetation (587 ng/kg), primarily as a result of direct air-to-leaf transfer. Concentrations of benzene in beef and milk are predicted to be low (<3 ng/kg) due to the low bioaccumulation potential of benzene. Predicted background levels of benzene in beef fat are about 300 times less than the measured concentration in human adipose tissue, which is in accordance with the general pattern observed by Travis *et al.* (1988).

Given that the food chain is not a major pathway of human exposure to benzene, we used direct biological sampling data to estimate human exposure to the general population. Three independent methods were used to estimate the background average daily intake of benzene by the general population of the United States. Two estimates based on measured concentrations of benzene in personal air and exhaled air yield intake estimates of 73 and 63 µg/day, respectively. These estimates based on biomonitoring agree well with our assessment of human exposure (72 µg/day) based on a pharmacokinetically derived estimate of steady-state benzene levels in human adipose tissue.

While inhalation is the primary route of human exposure to background levels of benzene in the environment, smoking is by far the largest anthropogenic source of background human exposure to benzene. Average smokers (20 cigarettes a day) take in about three times more benzene daily from smoking than from exposure to background benzene contamination. Since the increased lifetime risk associated with human exposure to background levels of benzene is  $1 \times 10^{-4}$ , we conclude that exposure to benzene may pose a potential health threat to the U.S. population.

## REFERENCES

- Agency for Toxic Substances and Disease Registry (ATSDR) (1987). "Toxicological Profile for Benzene" (Draft). U.S. Public Health Service, Atlanta.
- Allen, D. T., Cohen, Y., and Kaplan, I. R. (1990). "Intermedia Pollutant Transport: Modeling and Field Measurements". Plenum, New York.
- Antoine, S. R., DeLeon, I. R., and O'Dell-Smith, R. M. (1986). Environmentally significant volatile organic pollutants in human blood. *Bull. Environ. Contam. Toxicol.* 36, 364-371.
- Bidleman, T. F. (1988). Atmospheric processes. *Environ. Sci. Technol.* 22, 361-367.
- Bozzelli, J. W., and Kebbekus, B. B. (1982). A study of some aromatic and halocarbon vapors in the ambient atmosphere of New Jersey. *J. Environ. Sci. Health.* 17(5), 693-711.
- Bruggeman, W. A., Oppenhuizen, A., Wybenga, A., Hutzinger, O. (1984). Bioaccumulation of super-lipophilic chemicals in fish. *Toxicol. Environ. Chem.* 7, 173-189.

Chiou, C.  
Bio  
Cohen,  
ene  
Cross,  
of I  
opr  
Fishbe  
bor  
Geyer,  
cho  
Gilber  
As  
Er  
Hartw  
Sp  
old  
ro  
Hartw  
(I  
as  
Hatte  
hi  
Higgi  
p  
K  
Inter  
o  
V  
Intern  
F  
Lym  
r  
Mac  
t  
Mac  
Mac  
Mar  
Nat  
Pov  
Sau  
Sch  
SR  
Tr

2023380521

contaminants

a potentially  
silbert et al.,  
sure to ben-  
s high as 250  
d food items  
ever, show  
s than 1% of  
he U.S. The  
osed vegeta-  
concentrations  
e to the low  
of benzene in  
on in human  
ed by Travis.

sure to ben-  
posure to the  
ate the back-  
f the United  
c in personal  
tively. These  
f human ex-  
e of steady-

ground levels  
genic source  
(cigarettes a day)  
exposure to  
k associated  
we conclude  
U.S. popula-

ical Profile for  
1: Modeling and  
nificant volatile  
on vapors in the  
lation of super-

- Chiou, C. T., Freed, V. H., Schmedding, D. W., and Kohner, R. L. (1977). Partition coefficient and bioaccumulation of selected organic chemicals. *Environ. Sci. Technol.* 11, 473-478.
- Cohen Y., and Ryan, P. A. (1985). Multimedia modeling of environmental transport: Trichloroethylene test case. *Environ. Sci. Technol.* 19, 412-417.
- Cross, A. J., McFarlane, J. C., and Frank, C. W. (1979). "Benzene Vapor Depletion in the Presence of Plants." Environmental Monitoring and Support Laboratory, Office of Research and Development, U.S. Environmental Protection Agency, Las Vegas.
- Fishbein, L. (1984). An overview of environmental and toxicological aspects of aromatic hydrocarbons. I. Benzene. *Sci. Total Environ.* 40, 189-218.
- Geyer, H., Scheunert, I., and Korte, F. (1986). Bioconcentration potential of organic environmental chemicals in humans. *Regul. Toxicol. Pharmacol.* 6, 313-347.
- Gilbert, D., Byrne, M., Harris, J., Sieber, W., and Woodruff, C. (1982). "An Exposure and Risk Assessment for Benzene, Final Draft Report." Prepared by Arthur D. Little, Inc., for the U.S. Environmental Protection Agency, Office of Water and Waste Management, Washington, DC.
- Hartwell, T. D., Pellizzari, E. D., Perritt, R. L., Whitmore, R. W., Zelon, H. S., Sheldon, L. S., Sparacino, C. M., and Wallace, L. (1987a). Results from the total exposure assessment methodology (TEAM) study in selected communities in northern and southern California. *Atmos. Environ.* 21(9), 1995-2004.
- Hartwell, T. D., Pellizzari, E. D., Perritt, R. L., Whitmore, R. W., Zelon, H. S., and Wallace, L. (1987b). Comparison of volatile organic levels between sites and seasons for the total exposure assessment methodology (TEAM) study. *Atmos. Environ.* 21(11), 2413-2424.
- Hartemer-Frey, H. A., and Travis, C. C. (1989). Pentachlorophenol: Environmental partitioning and human exposure. *Arch. Environ. Contam. Toxicol.* 18, 482-489.
- Higgins, C. E., Griest, W. H., and Olerich, G. (1983). Application of tenax trapping to analysis of gas phase organic compounds in ultra-low tar cigarette smoke. *J. Assoc. Off. Anal. Chem.* 66, 1074-1083.
- International Agency for Research on Cancer (IARC). (1982). "IARC Monograph on the Evaluation of the Carcinogenic Risk of Chemicals to Humans: Some Industrial Chemicals and Dyestuffs," Vol. 29, pp. 93-148. Lyon, France.
- International Commission on Radiation Protection (ICRP). (1975). "Report of the Task Group on Reference Man," No. 23. Pergamon, New York.
- Lyman, J. W., Reehl, W. F., and Rosenblatt, D. H. (1982). "Handbook of Chemical Property Estimation Methods." McGraw-Hill, New York.
- Mackay, D., Paterson, S., Cheung, B., and Neely, W. B. (1985a). Evaluating the environmental behavior of chemicals with a level III fugacity model. *Chemosphere*, 14(3/4), 335-374.
- Mackay, D., Paterson, S., and Cheung, B. (1985b). Evaluating the environmental fate of chemicals: The fugacity-level III approach as applied to 2,3,7,8-TCDD. *Chemosphere*, 14, 859-863.
- Mackay, D. D., Paterson, S., and Schroeder, W. H. (1986). Model describing the rates of transfer processes of organic chemicals between atmosphere and water. *Environ. Sci. Technol.* 20(8), 810-816.
- Mars, S. J., and Lee, S. S. (1978). "Assessment of Human Exposure to Atmospheric Benzene." EPA-450/3-78-031. U.S. Environmental Protection Agency, Research Triangle Park, NC.
- National Research Council (NRC) (1980). "Drinking Water and Health," Vol 3, pp. 80-86, 261, 262. National Academy of Science Press, Washington DC.
- Powell, D. H., and Tucker, W. A. (1986). Nondrinking-water exposure. In "Benzene in Florida Groundwater: An Assessment of the Significance to Human Health." Florida Petroleum Council, Tallahassee, FL.
- Sauer, T. C., Jr. (1981). Volatile organic compounds in open ocean and coastal surface waters. *Org. Geochem.* 3, 91-101.
- Scheunert, I., Tepp, E., Schmitzer, J.U., Klein, W., and Korte, F. (1985). Formation and fate of bound residues of [<sup>14</sup>C]benzene and [<sup>14</sup>C]chlorobenzenes in soil and plants. *Ecotoxicol. Environ. Saf.* 9, 159-170.
- SRI International. (1988). "Directory of Chemical Producers U.S., 1987."
- Travis, C. C., and Arms, A. D. (1988). Bioconcentration of organics in beef, milk, and vegetation. *Environ. Sci. Technol.* 22(9), 271-274.

2023380522

- Travis, C. C., and Hattemer-Frey, H. A. (1992). Human exposure to 2,3,7,8-TCDD. *Chemosphere*, 16(10-12), 2321-2342.
- Travis, C. C., and Hattemer-Frey, H. A. (1990a). Concentration of TCDD in fish and the potential for human exposure. *Environ. Int.* 16(2), 153-162.
- Travis, C. C., and Hattemer-Frey, H. A. (1990b). Human exposure to dioxin. *Sci. Total Environ.*, in press.
- Travis, C. C., Craig, P. K., and Bowers, J. C. (1990a). Characterization of human exposure to ambient levels of benzene using pulmonary "wash-out" data. *Atmos. Environ.*, in press.
- Travis, C. C., Quillian, J. L., and Arms, A. D. (1990b). Pharmacokinetics of benzene. *Toxicol. Appl. Pharmacol.* 102, 400-420.
- Travis, C. C., Hattemer-Frey, H. A., and Arms, A. D. (1993). Relationship between dietary intake of organic chemicals and their concentrations in human adipose tissue and breast milk. *Arch. Environ. Contam. Toxicol.* 27, 473-478.
- Tucker, W. A., Huang, C., and Dickinson, R. E. (1986). Modeling transport of benzene in the subsurface. In "Benzene in Florida Groundwater: An Assessment of the Significance to Human Health." Florida Petroleum Council, Tallahassee, FL.
- U.S. Environmental Protection Agency (U.S. EPA). (1983). "Benzene: Occurrence in Drinking Water, Food, and Air." Sciences and Technology Branch, Criteria and Standards Division, Office of Drinking Water, Washington DC.
- U.S. Environmental Protection Agency (U.S. EPA). (1984). National emission standards for hazardous air pollutants: Regulation of benzene. *Fed. Reg.* 49(10), 23,478-23,493.
- U.S. Environmental Protection Agency (U.S. EPA). (1986a). "Superfund Public Health Evaluation Manual." EPA-540/1-86-010. Office of Emergency and Remedial Response, Washington, DC.
- U.S. Environmental Protection Agency (U.S. EPA). (1986b). "Broad Scan Analysis of the FY83 National Human Adipose Tissue Survey Specimens: Volatile Organic Compounds," Vol. 2. National Human Monitoring Program, Field Studies Branch, Design and Development Branch, Office of Toxic Substances, Washington, DC.
- Wakeman, S. C., Canuel, F. A., and Doering, P. H. (1986). Geochemistry of volatile organic compounds in groundwater: Multicolumn experiments with <sup>13</sup>C-model compounds. *Geochim. Cosmochim. Acta* 50, 1163-1172.
- Wallace, L. (1986). Personal exposures, indoor and outdoor air concentrations, and exhaled breath concentrations of selected volatile organic compounds measured for 600 residents of New Jersey, North Dakota, North Carolina and California. *Toxicol. Environ. Chem.* 12, 215-236.
- Wallace, L., Pelizzetti, E., Hartwell, T., Perritt, R., and Ziegelmajer, R. (1987). Exposure to benzene and other volatile compounds from active and passive smoking. *Am. J. Public Health* 77, 272-279.
- Wallace, L., Pelizzetti, E., Hartwell, T., Sparacino, C., Sheldon, L., and Zelom, M. (1985). Personal exposures, indoor-outdoor relationships and breath levels of toxic air pollutants measured for 352 persons in New Jersey. *Atmos. Environ.* 19, 1651-1661.
- Wallace, L., Zwidinger, R., Erickson, M., Cooper, S., Whitaker, D., and Pelizzetti, E. (1982). Monitoring individual exposure: Measurements of volatile organic compounds in breathing-zone air, drinking water, and exhaled breath. *Karpat. Rev.* 8, 269-282.
- Wetster, R. C., Malboeuf, H. I., Grunberg, L. D., and Craig, J. C. (1986). Benzene levels in ambient air and breath of smokers and nonsmokers in urban and pristine environments. *J. Toxicol. Environ. Health.* 18, 567-573.
- Yang, Y.-Y., and Nelson, C. R. (1986). An estimation of daily food usage factors for assessing radon dioxide intake in the U.S. population. *Health Phys.* 50(2), 245-257.

2023380523

March  
*Workshop on Indoor Air Quality*

NOTICE  
This material may be  
protected by copyright  
law (Title 17 U.S. Code).

## Major Sources of Exposure to Benzene and Other Volatile Organic Chemicals<sup>1,2</sup>

Lance Wallace<sup>3</sup>

Received March 7, 1989

The major sources of human exposure to about a dozen volatile organic chemicals (VOCs) have recently been identified.<sup>1</sup> For nearly every chemical, the major sources of exposure are completely different from the major sources of emissions. This finding implies that current environmental regulations and control strategies are misdirected. Important sources of exposure are typically not regulated in any way, whereas unimportant sources are heavily regulated. Vast sums of money are spent on problems involving little risk (e.g., hazardous waste sites), whereas few resources are expended on problems involving higher risk (e.g., indoor air pollution). The following paper summarizes recent findings regarding major sources of exposure to several VOCs. Benzene is selected as a case study. Brief discussions of tetrachloroethylene and paradichlorobenzene are also included.

**KEY WORDS:** Benzene; exposure; organic chemicals; tetrachloroethylene.

### 1. INTRODUCTION

About 800 people have now had their exposure to VOCs measured directly.<sup>(2-4)</sup> Since they were selected to represent a larger population of about a million residents of eight U.S. cities, their exposures probably represent the nationwide experience fairly. We review these findings, and consider their implications on the nation's environmental policies.

EPA's TEAM Studies targeted about 25-30 chemicals, of which about 15 were found to be prevalent (measurable in more than 20% of samples). Simultaneous sampling of personal air, outdoor air, and drinking water showed that, for most of the chemicals, inhalation provided 99% of the exposure. (Exceptions were chlo-

reform and the three other trihalomethanes, which have important routes through drinking water and beverages; and limonene, a food and beverage additive. Even for these exceptions, inhalation was an important if not dominant route of exposure.) Therefore, we shall concentrate on inhalation exposure in what follows. We shall consider three chemicals of interest: benzene, tetrachloroethylene, and paradichlorobenzene.

### 2. BENZENE

Exposures to benzene were measured for about 630 people in five locations. Outdoor air levels were measured near the homes of about 220 participants. The personal exposures averaged 16 µg/m<sup>3</sup>; the outdoor air levels averaged 6 µg/m<sup>3</sup> (Table I). If we assume that outdoor air infiltrates homes and workplaces with no losses as it crosses the building envelopes, then we can attribute no more than 6 µg/m<sup>3</sup> to outdoor sources: the remaining 10 µg/m<sup>3</sup> is due to some combination of personal activities and indoor sources.

<sup>1</sup> Paper presented at the Workshop on Exposure and Risk, Harvard University, Cambridge, Massachusetts, December 6-8, 1988.

<sup>2</sup> This paper has not been reviewed for policy implications by the U.S. Environmental Protection Agency and does not necessarily reflect EPA policy.

<sup>3</sup> U.S. Environmental Protection Agency, 401 M Street, SW, RD-480, Washington, D.C. 20460.

2023380524

**Table I. Personal Exposures to Benzene Compared to Ambient Levels in Five TEAM Study Locations**

Location	No. of Samples		Concentration <sup>a</sup>	
	Personal	Outdoor	Personal	Outdoor
NJ	340	86	28	9
MD	70	70	19	8
L.A.	232	132	14	8
A-P	68	10	8	2
NC	24	6	9	3
Total	734	304	16	6

<sup>a</sup> Population-weighted 24-hour arithmetic mean ( $\mu\text{g}/\text{m}^3$ ): NJ = Bayonne-Elizabeth, New Jersey (Fall 1981); MD = Baltimore, Maryland (Spring 1987); L.A. = Los Angeles, California (two seasons, 1984 and 1987); A-P = Antioch-Pittsburgh, California (June 1984); NC = Greensboro, North Carolina (May 1982).

### 2.1. Active Smoking

Before discussing what these sources are, however, we need to take account of a source that is *not* reflected in the average measured exposure of  $16 \mu\text{g}/\text{m}^3$ : mainstream cigarette smoke. Based on measurements of benzene in mainstream cigarette smoke<sup>(7)</sup>, Wallace<sup>(1,8)</sup> has estimated that mainstream smoke contributes about  $1.8 \mu\text{g}/\text{day}$  to the average smoker's intake of benzene. This corresponds to an average *additional* exposure for smokers of about  $90 \mu\text{g}/\text{m}^3$  (assuming  $20 \text{ m}^3/\text{day}$  respiration rate). Since there are about 50 million smokers in the U.S., the total benzene exposure for them is roughly equal to the total benzene exposure from all other sources for the remainder of the population.

### 2.2. Passive Smoking

Studies of 500 homes in both the U.S.<sup>(9)</sup> and in West Germany<sup>(10)</sup> have indicated that homes with smokers have median indoor air benzene concentrations about  $4 \mu\text{g}/\text{m}^3$  higher than in homes without smokers. Since at least half of U.S. homes contain smokers, we can calculate that about  $2 \mu\text{g}/\text{m}^3$  is contributed, on average, by passive smoking.

### 2.3. Automobile Travel

A recent study in California (D. Shikiya, unpublished data) indicates that automobile interior concentrations of benzene during commutes in Los Angeles average about 13 ppb ( $40 \mu\text{g}/\text{m}^3$ ). The same concentration had been estimated by Wallace,<sup>(8)</sup> based on the TEAM Stud-

ies in California. Assuming these levels are applicable to the rest of the country, and assuming about 1 hour per day in the automobile, this exposure would contribute roughly another  $2 \mu\text{g}/\text{m}^3$  to average exposure. Besides traveling in autos, filling gas tanks could contribute a portion of benzene exposure, although the total estimated contribution is only  $0.2 \mu\text{g}/\text{m}^3$  (about 10% of the effect of automobile travel).

### 2.4 Attached Garages

Gammage<sup>(11)</sup> and McClenney<sup>(12)</sup> reported finding gasoline vapor in homes with attached garages. This could arise from evaporative emissions following parking, or from storage of gasoline in the garage. No estimates of the extent of exposure from these sources have yet been made.

### 2.5 Products and Materials

More than 200 products and materials were found to emit benzene in studies carried out by NASA.<sup>(13)</sup> Sheldon *et al.*,<sup>(14,15)</sup> found benzene being emitted from several paints and adhesives, although indoor concentrations in two buildings constructed from these materials were not elevated. Thus these sources may contribute to exposure, although no estimates have been made of the contribution of products and materials to personal exposure.

### 2.6. Occupational

Workers in the chemical, manufacturing, and transportation industries may be exposed to elevated levels of benzene. However, the contribution of occupational exposure to the average exposure measured in the TEAM Studies appears to be relatively small.

### 2.7. Outdoor Air

A study of morning rush-hour (6-9 AM) concentrations of benzene in 39 cities gave a median concentration of  $6 \mu\text{g}/\text{m}^3$ ,<sup>(16)</sup> agreeing well with the mean value of  $6 \mu\text{g}/\text{m}^3$  observed over 24-hr periods in residential areas in the TEAM Study. The major sources of benzene in the atmosphere are mobile sources (auto exhaust and evaporative emissions) and industrial (petroleum refineries, petrochemical manufacturing, coke ovens) sources. Mobile sources appear to be more important than sta-

2023380525

tionary sources in contributing to outdoor benzene levels. For example, in the 39-city study, cities with heavy petrochemical industries such as Houston, Texas (ranked 7th); Beaumont, Texas (17th); Lake Charles, Louisiana (19th); and Orange, Texas (31st) were not particularly elevated in benzene concentrations. Recent estimates of emissions attribute about 85% of emissions to mobile sources, 15% to stationary sources.

### 2.8 Residence Near Industry

The first TEAM Study<sup>(3)</sup> found no difference in benzene exposures of 150 subjects living within 1 km of chemical and petroleum refineries in Bayonne and Elizabeth, New Jersey, compared to 150 subjects living more than 1 km distant. Since such facilities are concentrated in only a few places in the U.S., even a positive finding would have little effect on the nationwide average exposure to benzene.

### 2.9. Food

Although a number of publications have referred to benzene in vegetables, meat, and eggs, the TEAM Study found little evidence that diet made any difference in benzene levels in breath. Since the same breath measurements were conclusive in identifying smoking as an important source, it is felt that food cannot be an important source of exposure to benzene.

### 2.10. Wood Smoke

Few measurements are available to allow an estimate of the importance of wood smoke on benzene exposure. Since only a few localities use wood burning to an appreciable extent, and then for only a few months of the year, wood smoke should not make an appreciable contribution to nationwide average exposure to benzene.

### 2.11. Summary: Sources of Benzene Exposure

From the above considerations, we can construct a nationwide benzene exposure budget (Table II) apportioning the observed benzene exposures to the most important sources. The results indicate that smoking accounts for roughly half of the exposure, with the remaining half split fairly evenly between personal activities ( $\sim 30\%$ ) and the traditional outdoor sources ( $\sim 20\%$ ).

On the other hand, emissions present a very differ-

Table II. Benzene "Exposure Budget": Major Sources of Benzene Exposures and Risks

Activity	Intake ( $\mu\text{g}/\text{day}$ )	Pop. at risk ( $\times 10^6$ )	Total risk (%)
Smoking	1800	53	50
Unknown personal	150	240	20
Ambient	120	240	20
Passive smoking	50	190	5
Occupational	10000	0.25	1
Filling gas tank	10	100	<1

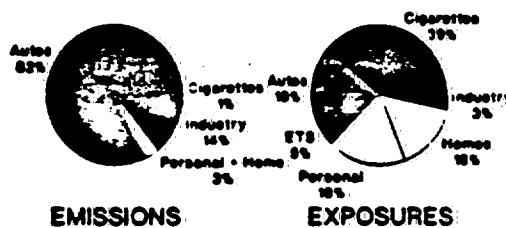


Fig. 1. Benzene: Emissions vs. exposures (TEAM study, Los Angeles, 1987).

ent picture. The traditional sources—motor vehicles and industry—account for 97% of the total emissions, compared to 3% from cigarettes and materials. The relative importance of these different sources are compared for emissions and exposures in Fig. 1.

These findings have important effects on our regulatory and control strategies. For example, if emissions from all stationary sources were reduced by a Draconian 50%, the total reduction in population exposure would be an unnoticeable 2% ( $50\% \times 15\% \times 20\%$ ). The same effect could be achieved by reducing the average benzene content of cigarettes by 4% (from 57 to 55  $\mu\text{g}/\text{m}^3$ ). This raises the interesting question of whether a steel company might trade "exposure credits" with a tobacco company—the latter reducing the tar and nicotine content of its cigarette slightly (in return for appropriate remuneration) to allow a below-standard coke oven to continue in operation. The idea of trading in exposure rather than emissions is described in Smith.<sup>(17)</sup>

### 3. TETRACHLOROETHYLENE

Exposures to tetrachloroethylene are compared to outdoor levels in Table III. The difference between per-

2023380526

Table III. Personal Exposures to Tetrachloroethylene Compared to Ambient Levels in Six TEAM Study Locations

Location	No. of Samples	Concentration*		
	Personal	Outdoor	Personal	Outdoor
NJ	539	155	28	5
MD	70	70	7	2
LA	232	131	14	4.5
A-P	76	10	6	0.6
ND	23 <sup>b</sup>	6	9	1
NC	24	6	7	0.9
Total	964	378	12	2.3

\* Population-weighted 24-hr arithmetic mean ( $\mu\text{g}/\text{m}^3$ ). NJ = Bayonne-Elizabeth, New Jersey (three seasons, 1981-83); MD = Baltimore, Maryland (Spring 1987), L.A. = Los Angeles, California (two seasons, 1984 and 1987); A-P = Antioch-Pittsburg, California (June 1984); ND = Devils Lake, North Dakota (October 1982); NC = Greensboro, North Carolina (May 1982).

<sup>b</sup> One outlier ( $800 \mu\text{g}/\text{m}^3$ ) removed.

sonal exposures and outdoor concentrations is even more striking than for benzene, with outdoor air providing only about 20% of total exposure. Unlike benzene, however, tetrachloroethylene has few sources of exposure. The main source of exposure for most people is probably dry-cleaned clothes.

### 3.1 Dry-Cleaned Clothes

Early TEAM studies showed that tetrachloroethylene levels were higher among employed people, suggesting that exposure to one's own or to coworkers' dry-cleaned clothes could be important. A recent TEAM study<sup>(18)</sup> has indicated that tetrachloroethylene levels in homes increase by factors of 100-fold (to levels exceeding  $100 \mu\text{g}/\text{m}^3$ ) following the introduction of dry-cleaned clothes into the home. (The study also indicated that indoor air levels decrease when the clothes are removed from the home and increase when they are put back, thus supporting the notion that "airing out" the clothes on a balcony or patio before introducing them into the home can be effective in reducing exposure.) The same study showed that wearing the clothes also increased personal exposure. Finally, a small but noticeable source of exposure occurs during the few minutes the clothes are being picked up at the dry cleaning shop; earlier TEAM Studies<sup>(19)</sup> indicated that levels in dry-cleaning shops varied between 10,000 and 20,000  $\mu\text{g}/\text{m}^3$ . Thus a 5-min exposure would provide as much tetrachloroethylene as 5 days of normal exposure. The contributions to total exposure of these four sources (the home, the

office, the dry-cleaning shop, and the outdoors) are assessed in Table IV.

### 3.2. Outdoor Air

The main use of tetrachloroethylene is as a dry-cleaning solvent—a majority of U.S. dry-cleaning shops employ tetrachloroethylene as the primary solvent. Thus the dry-cleaning shop is considered the major source of outdoor tetrachloroethylene. However, these emissions account for no more than 20% of total exposure. Thus, reducing emissions from dry cleaning shops by our unrealistic factor of 50% would result in a barely noticeable 10% reduction in exposure. The same reduction might be achievable if people hung their dry cleaning outside for an 8-hr period before taking it into the house. The major sources of exposure are compared to the major emission sources in Fig. 2.

## 4. PARADICHLOROBENZENE

Results from six TEAM Study cities showed that paradichlorobenzene was almost exclusively an indoor air pollutant, outweighing outdoor air by more than 20

Table IV. National Exposure Budget for Tetrachloroethylene

Source category	Population exposed (000000)	Mean exposure ( $\mu\text{g}/\text{m}^3$ )	Percent of time exposed	Contribution to total exposure ( $\mu\text{g}/\text{m}^3$ )
Office	120	18	22	2
Home	238	6	83	5
Outdoors	238	3	100	3
Dry-cleaning shop	85	10,000	0.01	<1
Other uses	?	?	?	=1
Total	238	12	100	12

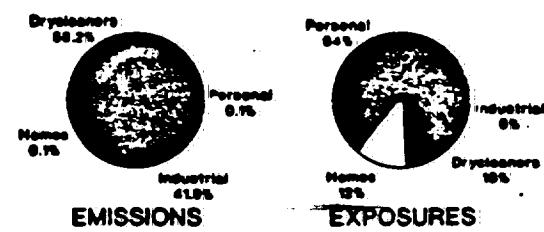


Fig. 2. Tetrachloroethylene: Emissions vs. exposures (TEAM study, Los Angeles, 1987).

2023380527

to 1 (Table V). Assuming that one third of homes contain p-DCB, we may calculate that users of these products are increasing their exposures by factors of roughly 60 compared to nonusers.

#### 4.1. Sources of Exposure

This chemical has two major uses: to mask odors and to kill moths. Both uses require that the chemical maintain a high concentration in the home for periods of months or even years. A large number of American homes may contain high levels of p-DCB. Many schools, offices, hotels and other places with public restrooms also use p-DCB to mask odors.

About 12 million pounds annually is used to kill moths. An estimated 25% of American households contain mothballs, moth crystals, or moth cakes formed from nearly pure p-DCB, although only 12% of TEAM Study homes in Baltimore and Los Angeles reported having moth repellents in their homes.

About 70% of TEAM Study homes in Baltimore and Los Angeles reported using air fresheners or bathroom deodorants. Paradichlorobenzene accounts for a fraction of the air freshener market (perhaps 10%). Assuming 25% of homes have p-DCB moth repellents and an additional 7% have p-DCB air fresheners, we may calculate that about a third of the 85 million homes in the U.S. contain p-DCB.

In 1986, following a two-year test of male and female rats and mice, the National Toxicology Program announced that p-DCB caused several different types of

malignant tumors in both sexes of the mice and in male rats<sup>(20)</sup>. Traditionally, when a chemical causes cancer in two different species of mammals, it is considered a probable human carcinogen. In this case, because the tumors occurred in the male rat kidney and the mouse liver, both of which have been questioned for their relevance to human cancer, p-DCB has been provisionally classified as a possible human carcinogen.

#### 5. DISCUSSION

For each of the three chemicals discussed above, the "traditional" sources of emissions (mobile sources, industry) have accounted for only 2-20% of total human exposure. This same conclusion has been documented for a number of other volatile organic chemicals: styrene, xylenes, ethylbenzene, trimethylbenzenes, chloroform, trichloroethylene,  $\alpha$ -pinene, limonene, decane, undecane, etc.<sup>(1,2)</sup> For most of these chemicals, the major sources of exposure have been identified (personal activities, consumer products, building materials), but cannot be regulated under existing environmental authorities.

This situation has led to a peculiar split in the perception of risk. The public perceives indoor air pollution as considerably less risky than, say, hazardous waste sites (*Environment*, August 1988) whereas experts at EPA put indoor air and consumer product exposure at the top of the list of health risks, with hazardous waste sites near the bottom.<sup>(21)</sup> Nonetheless, the amount of resources devoted to these two problems reflect the public perception, not that of the experts.

How can this situation be rectified? A continuing process of consumer information and media attention may ultimately result in greater public awareness of the problem. Some steps to reduce exposure can be taken by the public without waiting for cumbersome government attempts at regulation. Other actions, such as setting up consensus guidelines, can be taken by professional organizations: e.g., ASHRAE (ventilation requirements), ASTM (standardized testing for organic emissions from building materials). Information on the economic impacts of indoor air pollution may ultimately convince employers to improve their employees' working conditions. Market forces may also play a role—manufacturers may find substitute chemicals or processes leaving less residue in their products, if the public demands it. (The bellwether chemical here may be formaldehyde—particleboard with up to ten times less formaldehyde emission potential is now available, at a price).

Table V. Personal Exposures to Para-dichlorobenzene Compared to Ambient Levels in Six TEAM Study Locations

Location	No. of Samples		Concentration <sup>a</sup>	
	Personal	Outdoor	Personal	Outdoor
NJ	539	155	55	1.3
MD	70	70	33	— <sup>b</sup>
L.A.	232	131	15	1.6
A-P	76	10	6	0.3
ND	24	6	16	0.7
NC	24	6	11	0.7
Total	963	378	23	1

<sup>a</sup> Population-weighted 24-hour arithmetic mean ( $\mu\text{g}/\text{m}^3$ ). NJ = Bayonne-Elizabeth, New Jersey (three seasons, 1981-83); MD = Baltimore, Maryland (Spring 1987); L.A. = Los Angeles, California (two seasons, 1984 and 1987); A-P = Antioch-Pittsburg, California (June 1984); ND = Devils Lake, North Dakota (October 1982); NC = Greensboro, North Carolina (May 1982).

<sup>b</sup> Not measured using Tenex.

2023380528

## 6. CONCLUSIONS

We have shown that the major sources of emissions of three chemicals account for only a small proportion (2-20%) of total exposure. For all three chemicals (and for a number of other VOCs), the main sources of exposure are personal activities (such as smoking or wearing dry-cleaned clothes) or consumer products (such as room air fresheners). These sources of exposure are intrinsically more difficult to regulate than the traditional mobile and industrial sources; moreover, authority to regulate them appears to be lacking in many instances. A combination of public information, voluntary consensus guidelines, and market forces are needed to deal with this problem.

## REFERENCES

1. L. A. Wallace (1987). *Total Exposure Assessment Methodology (TEAM) Study: Summary and Analysis, Vol. I* (Washington, D.C., United States Environmental Protection Agency).
2. R. W. Handy, et al. (1987). *Total Exposure Assessment Methodology (TEAM) study: Standard Operating Procedures, Vol. IV* (Washington, D.C., United States Environmental Protection Agency).
3. E. D. Pellizzari, K. Perritt, T. D. Hartwell, L. C. Michael, R. Whitmore, R. W. Handy, D. Smith, and H. Zelon (1987). *Total Exposure Assessment Methodology (TEAM) study: Elizabeth and Bayonne, New Jersey; Devils Lake, North Dakota; and Greensboro, North Carolina, Vol. II* (Washington, D.C. United States Environmental Protection Agency).
4. E. D. Pellizzari, K. Perritt, T. D. Hartwell, L. C. Michael, R. Whitmore, R. W. Handy, D. Smith, and H. Zelon (1987). *Total Exposure Assessment Methodology (TEAM) Study: Selected Communities in Northern and Southern California, Vol. III* (Washington, D.C. United States Environmental Protection Agency).
5. L. A. Wallace, E. D. Pellizzari, T. D. Hartwell, C. Sparacino, R. Whitmore, L. Sheldon, H. Zelon, and R. Perritt (1987). "The TEAM Study: Personal Exposures to Toxic Substances in Air, Drinking Water, and Breath of 400 Residents of New Jersey, North Carolina, and North Dakota." *Environ. Res.* 43; 290-307.
6. L. A. Wallace, E. D. Pellizzari, T. D. Hartwell, R. Whitmore, H. Zelon, R. Perritt, and L. Sheldon (1988). "The California TEAM Study: Breath Concentrations and Personal Exposures to 26 Volatile Compounds in Air and Drinking Water of 188 Residents of Los Angeles, Antioch, and Pittsburg, CA;" *Aerosol Environ.* 22, 2141-2163.
7. C. Higgins et al. (1983). "Applications of Tenax Trapping to Cigarette Smoking," *J. Assoc. Official Analytical Chem.* 66, 1074-1083.
8. L. A. Wallace (1989). "Major Sources of Benzene Exposure," *Env. Health Perspectives* 82, 165-169.
9. L. Wallace, E. Pellizzari, T. Hartwell, K. Perritt, and R. Ziegenfus, (1987). "Exposures to Benzene and Other Volatile Compounds from Active and Passive Smoking," *Arch. Environ. Health* 42, 272-279.
10. C. Krause, W. Mailaha, R. Nagel, C. Schulz, B. Seifert, and D. Ullrich (1987). "Occurrence of volatile organic compounds in the air of 500 Homes in the Federal Republic of Germany," in *Proceedings of The 4th International Conference on Indoor Air Quality and Climate*, Vol. 1, (Institute for Soil, Water, and Air Hygiene, Berlin ), pp. 102-106.
11. R. B. Gammie, D. A. White, and K. C. Gupta, (1984). "Residential Measurements of High Volatility Organics and Their Sources," in *Indoor Air*, v. 4, (Swedish Council for Building Research, Stockholm, Sweden), pp. 157-162.
12. W. A. McCleary, T. A. Lumpkin, J. D. Pleil, K. D. Oliver, D. K. Bubacz, J. W. Faircloth, and W. H. Daniels, (1986). "Canister-based VOC Samplers," in *Proceedings of the EPA/APCA Symposium on Measurement of Toxic Air Pollutants* (Air Poll. Cont. Assoc. Pittsburgh, Pennsylvania).
13. National Aeronautics and Space Industry, Houston, Texas. Materials Testing Data Base.
14. L. Sheldon, R. Handy, T. D. Hartwell, R. W. Whitmore, H. S. Zelon, and E. D. Pellizzari (1988). *Indoor Air Quality in Public Buildings, Volume I* (U.S. Environmental Protection Agency, Washington, D.C., EPA/600/6-88/009a).
15. L. Sheldon, H. Zelon, J. Sickles, C. Eaton, and T. Hartwell (1988). *Indoor Air Quality in Public Buildings, Volume II* (U.S. Environmental Protection Agency, Washington D.C., EPA/600/6-88/009b).
16. R.L. Seils (1987). "June-September, 6-9 AM, Ambient Air Benzene Concentrations in 39 U.S. Cities, 1984-1986," in *Proceedings of the 1987 U.S. EPA-APCA Symposium on Measurement of Toxic and Related Air Pollutants*, Air Pollution Control Association, Pittsburgh, Pennsylvania.
17. K. Smith (1988). "Air Pollution: Assessing Total Exposure in the United States," *Environment* 30, 10-38.
18. E. D. Pellizzari, K. W. Thomas, D. J. Smith, R. Perritt, and M. Morgan, (1987). *Total Exposure Assessment Methodology (TEAM): 1987 Study in New Jersey Final Draft* (Contract # 68-02-4544. U.S. Environmental Protection Agency, Washington D.C.).
19. E. D. Pellizzari et al. (1984). *Total Exposure Assessment Methodology (TEAM): Dry Cleaners Study* (EPA contract no. 68-02-3626, U.S. EPA, Office of Research and Development, Washington, D.C.).
20. National Toxicology Program (NTP) (1986). Technical Report on the Toxicity and Carcinogenesis of 1,4-Dichlorobenzene (CAS #106-46-7) in F344/n Rats and B6C3F1 Mice (Gavage Study). NTP Technical Report No. 319, Board Draft, March 1986.
21. U.S. Environmental Protection Agency (1988). *The Inside Story: A Consumer's Guide to Indoor Air Pollution* (Washington, D.C.).

2023380529

## QUESTIONNAIRE ASSESSMENT OF LIFETIME AND RECENT EXPOSURE TO ENVIRONMENTAL TOBACCO SMOKE

DAVID B. COULTAS,<sup>1,2</sup> GLENN T. PEAKE,<sup>1,2</sup> AND JONATHAN M. SAMET<sup>1,2</sup>

Coultas, D. B. (New Mexico Tumor Registry, Cancer Center, U. of New Mexico Medical Center, Albuquerque, NM 87131), G. T. Peake, and J. M. Samet: Questionnaire assessment of lifetime and recent exposure to environmental tobacco smoke. *Am J Epidemiol* 1989;130:338-47.

In a sample of 149 adult nonsmokers recruited in New Mexico in 1986, the authors assessed the reliability of questionnaire responses on lifetime exposure to tobacco smoke in the home. They also compared urinary cotinine levels with questionnaire reports of environmental tobacco smoke exposure during the previous 24 hours. The agreement of responses obtained on two occasions within six months was high for parental smoking during childhood: 84% for the mother and 83% for the father. For the amounts smoked by the mother and the father during the subject's childhood, the agreement between the two interviews was moderate: 52% and 39%, respectively. For the number of hours per day that each parent smoked in the home during the subject's childhood, the Spearman correlation coefficients also indicated only moderate reliability ( $r = 0.18$  for maternal smoking and  $r = 0.54$  for paternal smoking). For each set of interviews, responses concerning recent tobacco smoke exposure and urinary cotinine levels were correlated to only a modest degree. The authors conclude that adults can reliably report whether household members smoked during their childhood, but information on quantitative aspects of smoking is reported less reliably.

pyrrolidinones; questionnaires; tobacco smoke pollution

The term "passive smoking" refers to the involuntary exposure of nonsmokers to the combination of tobacco combustion products released by the burning cigarette and smoke components exhaled by the active smoker (1, 2). The adverse health effects of passive smoking on children and adults have been described in numerous epidemi-

ologic investigations (1, 2). However, despite the evidence linking malignant and nonmalignant diseases with active and passive smoking, tobacco smoking remains highly prevalent worldwide (1). In the United States at present, about 30 per cent of adults are active cigarette smokers (3), so that a large proportion of nonsmokers

Received for publication March 28, 1988, and in final form October 11, 1988.

<sup>1</sup> New Mexico Tumor Registry, Cancer Center, University of New Mexico Medical Center, Albuquerque, NM.

<sup>2</sup> Departments of Medicine and of Family, Community, and Emergency Medicine, and the Interdepartmental Program in Epidemiology, University of New Mexico, Albuquerque, NM.

<sup>3</sup> Department of Medicine, University of New Mexico, Albuquerque, NM.

\* Deceased.

Reprint requests to Dr. David B. Coultas, New Mexico Tumor Registry, Cancer Center, University of

New Mexico Medical Center, 800 Camino de Salud NE, Albuquerque, NM 87131.

Supported by Grant EPA CR811650 from the Environmental Protection Agency.

Dr. Coultas is a recipient of an Edward Livingston Trudeau Scholar Award from the American Lung Association.

The authors thank Dr. Helen Van Yunakis for providing the reagents for the radioimmunoassay and Irene Walkiw for technical assistance in performing the assays. Special thanks to the interviewers and to Lee Fernando, Rita Elliott, and Rebecca Musher for their help in preparing the manuscript.

2023380530

in this country are involuntarily exposed to environmental tobacco smoke (1, 2).

Although some health effects of passive smoking have been convincingly demonstrated, many questions on the health effects of passive smoking remain unanswered. More precise description of exposure-response relations is needed for assessment of the adverse effects on children and the development of lung cancer. Additionally, further studies on exposure to environmental tobacco smoke in the workplace are warranted because of the high prevalence of smoking among adults and public concern about this source of exposure. In most epidemiologic studies on involuntary smoking published to date, exposure has been assessed with questionnaires; for the purposes of some investigations, the questionnaires have spanned the entire lifespans of the subjects. Questionnaires will remain the most feasible method for assessing exposure to environmental tobacco smoke in new studies. However, the reliability and validity of questionnaire measures of involuntary smoking have not been adequately characterized.

In this study, we have assessed the reliability of a comprehensive questionnaire on lifetime exposure to environmental tobacco smoke in 149 adult nonsmokers. While validity is also of interest, no appropriate standard for comparison is available for a lifetime history. Questionnaire responses with poor reliability are also likely to have poor validity. In this sample, we also examined the relation between reports of recent exposure to environmental tobacco smoke and urinary cotinine levels.

#### MATERIALS AND METHODS

##### *Sample selection*

Between February and December of 1986, nonsmokers aged 18 years and older were recruited from Albuquerque, New Mexico, and the surrounding communities. Recruitment was accomplished by two methods: advertisements and direct contact with subjects from a population survey (4). In both approaches, we asked for volunteers

to participate in a study of indoor air quality that involved completing a questionnaire on two occasions and providing saliva and urine samples. The subjects were not informed that the study was directed specifically at exposure to environmental tobacco smoke. We attempted to stratify the sample uniformly by age and by sex but were not completely successful (table 1). Of our sample, 62 per cent were female, and only five males were aged 60 years and older.

##### *Data collection*

A structured questionnaire on lifetime and recent exposure to environmental tobacco smoke was administered by a trained interviewer to each subject on two occasions separated by approximately four to six months. Training involved familiarization and practice with the questionnaire and review of probing techniques, which were standardized. The interviews were conducted by four interviewers who completed 89.2, 5.4, 2.7, and 2.7 per cent of the first interviews and 38.2, 6.7, 54.4, and 0.7 per cent of the second interviews, respectively. We asked whether the subject's mother had smoked while pregnant with the subject, and we determined the smoking status of parents, spouses, and others from questions on whether these persons had smoked in the subject's home on a daily basis for six months or more. These questions referred to two time periods: birth to age 18 years and age 19 years to the time of the interview. These time periods were chosen to correspond to the usual ages for

TABLE I  
Age and sex distribution of 149 participants in a study of involuntary exposure to tobacco smoke,  
New Mexico, 1986

Age (years)	Males		Females	
	No.	%	No.	%
20-29	12	21.4	17	18.3
30-39	20	35.7	27	29.0
40-49	9	16.1	15	16.1
50-59	10	17.9	13	13.1
≥60	5	8.9	19	20.4

2023380531

living in the parents' home and subsequently living outside the parents' home. In addition, for each smoker, we asked about the type(s) of tobacco smoked (cigarette, pipe, or cigar), the amount of each type smoked in the home, the number of years each type was smoked, and the number of hours of exposure per day to each type in the home. Another set of questions asked about the amount of exposure during the previous 24 hours. The questions covered the number of smokers to which the subject was exposed, the type(s) of tobacco smoked (cigarette, pipe, or cigar), and the number of hours of exposure. These questions were asked separately for exposures at home, at work, in vehicles, and at social gatherings. At the time of the interview, a urine specimen was collected and frozen at -20°C until the cotinine assays were performed.

#### Cotinine assay

Cotinine was quantitated by a double antibody radioimmunoassay, as described by Langone et al. (5). A specific antiserum produced in rabbits was supplied by Dr. Helen Van Vunakis of Brandeis University (Waltham, MA). Urine samples were diluted 1:4 for the assay. The sensitivity of the assay in our hands was 36 pg/tube or 0.78 ng/ml of urine (4,204 pmol/liter). Urinary creatinine concentrations were determined by the Jaffe reaction (6), and the cotinine concentrations were standardized to the creatinine concentrations. Assays were performed without knowledge of questionnaire responses.

#### Data analysis

Reliability was assessed by comparison of the two lifetime histories for the exposure variables during the two time periods, birth to age 18 years and age 19 years to the time of the interview. Because of the small number of pipe and cigar smokers among parents ( $n = 24$ ) and spouses ( $n = 4$ ), we restricted our analysis to cigarette smokers. We summarized the per cent

agreement between the first and second interviews for categorical variables, which included mother's smoking during pregnancy; mother's, father's, and spouse's cigarette smoking status; amount smoked, categorized as less than one pack per day, one pack per day, and more than one pack per day; and number of other cigarette smokers in the household, categorized as none, one, and two or more. To discount chance agreements between the first and second interviews, Cohen's kappa was calculated for all categorical items and tested for significance (7, 8). Spearman rank order correlation coefficients (9) were calculated for continuous variables, which included both the number of years and the number of hours per day that the subject's mother, father, spouse, and others had smoked.

For questions on exposure to tobacco smoke during the previous 24 hours, we created summary variables for cigarette smoke exposure only, because exposure to pipe and cigar smokers was infrequent. The summary variables for cigarette smoke exposure included the total number of hours of exposure and the total number of cigarette smokers in all locations. To examine the relation between measures of short term exposure to environmental tobacco smoke within and between interviews, we calculated Spearman rank order correlations (9).

Data analyses were performed with standard programs of the Statistical Analysis System (10).

#### RESULTS

Of the 158 subjects enrolled for the first interview, 149 (94 per cent) also completed the second interview. Of the nine subjects who were not reinterviewed, there were seven males and two females, with mean ages of 43.6 years and 43.0 years, respectively. This report is based on responses of those 149 subjects who were reinterviewed. The age range of the 149 subjects was 21-79 years (mean = 43 years); 37.6 per cent were males and 62.4 per cent were females (table 1). The median duration between

2023380  
0132

interviews was 17 weeks, with a range of 6-35 weeks.

For the period birth to age 18 years, agreement between the first and second interviews was high for parental smoking status during childhood (table 2). The per cent agreement was similar for mother's and father's smoking during childhood and was lowest for maternal smoking during pregnancy. The percentage of unknown responses was highest for maternal smoking during pregnancy. The per cent agreement

and kappa statistic for the number of other cigarette smokers in the home during childhood were 77.0 per cent and 0.47 ( $p < 0.0001$ ), respectively.

In contrast to the high reliability of responses about parental smoking status during childhood, concordance was low for responses about the usual amount smoked in the home by the parents during childhood (table 3). The concordance was highest for the amount smoked by the mother and lowest for the amount smoked by the fa-

TABLE 2  
Percentage of nonsmokers reporting exposure to parental cigarette smoking during childhood, New Mexico, 1986

Response	Maternal smoking during pregnancy (n = 149)	Maternal smoking during childhood (n = 149)	Paternal smoking during childhood (n = 149)
<b>Yes</b>			
First interview	20.1	36.9	55.7
Second interview	20.1	32.9	56.4
<b>No</b>			
First interview	67.1	62.4	43.6
Second interview	64.4	67.1	42.9
<b>Unknown</b>			
First interview	12.8	0.7	0.7
Second interview	15.5	0.0	0.7
<b>Agreement</b>			
Concordance	85.9	94.0	92.6
Kappa	0.73*	0.87*	0.85*

\*  $p < 0.0001$ .

TABLE 3  
Percentage of nonsmokers reporting exposure to various amounts of cigarettes smoked by the parents during childhood and by the spouse during adulthood, New Mexico, 1986

Amount smoked	Maternal smoking during childhood (n = 68)	Paternal smoking during childhood (n = 79)	Spousal smoking during adulthood (n = 64)
<b>Less than one pack/day</b>			
First interview	62.5	70.9	84.4
Second interview	50.0	35.4	40.6
<b>One pack/day</b>			
First interview	20.8	11.4	7.8
Second interview	22.9	32.9	31.3
<b>More than one pack/day</b>			
First interview	6.3	10.1	6.3
Second interview	18.7	22.8	28.1
<b>Unknown</b>			
First interview	10.4	7.8	1.6
Second interview	10.4	8.9	0.0
<b>Agreement</b>			
Concordance	52.1	39.3	43.8
Kappa	0.22*	0.04*	-0.04*

\*  $p > 0.05$ .

2023380533

ther. Compared with the first interview, the percentage of subjects reporting parental smoking of one pack per day or more was higher at the second interview.

We also examined the reliability of responses on smoking status and amount smoked by sex and by age. The findings were similar to the overall analysis within strata defined by either sex or age, above and below age 40 years.

Spearman correlations were used to describe the agreement between the first and second interviews on the reported number of years and hours per day of exposure to environmental tobacco smoke during childhood. The correlation coefficients were high for responses on the number of years the parents and other smokers in the household had smoked (table 4). However,

for responses on the number of hours per day of smoke exposure in the home, the correlation coefficients were much lower (table 4).

We next examined the reliability of reported smoke exposure during adulthood (tables 3 and 5). After age 18 years, the numbers of subjects living with either their mother ( $n = 8$ ) or their father ( $n = 9$ ) were small. For this small group of subjects, the concordance of responses on parental smoking status was 100 per cent. Similarly, the per cent agreement on spouse's smoking status, as obtained at the two interviews, was 100 per cent ( $n = 67$ ). For the amount currently smoked by the spouse, the concordance was lower (table 3). Agreement between responses about the number of other cigarette smokers in the household

TABLE 4  
Mean years and hours per day of childhood cigarette smoke exposure reported by nonsmokers,  
New Mexico, 1986

Exposure variable	No.	First interview	Second interview	Spearman's $\rho$
Maternal smoking:				
Years*	33	15.4	15.7	0.76
Hours/day†	31	5.0	6.4	0.18
Paternal smoking:				
Years	57	16.1	15.4	0.75
Hours/day	55	4.8	4.8	0.54
Other household members' smoking:				
Years	26	13.9	13.2	0.63
Hours/day	20	9.2	8.4	0.51

\* "During the period from birth to age 18 years, for how many years did he/she smoke cigarettes?"

† "On average, during the period from birth to age 18 years, for how many hours per day were you exposed to individuals' cigarette smoke?"

TABLE 5  
Mean years and hours per day of adulthood cigarette smoke exposure reported by nonsmokers,  
New Mexico, 1986

Exposure variable	No.	First interview	Second interview	Spearman's $\rho$
Spouse's smoking:				
Years*	40	16.2	16.4	0.96
Hours/day†	39	5.9	5.5	0.23
Other household members' smoking:				
Years	67	8.3	8.2	0.78
Hours/day	58	12.7	10.3	0.54

\* "For how many years did he/she smoke cigarettes while you were sharing your home?"

† "On average, how many hours per day were you exposed to their cigarette smoke?"

2023380534

was 74.0 per cent ( $n = 66$ ), with a kappa value of 0.50 ( $p < 0.0001$ ).

Correlations between responses at the two interviews were high for the number of years the spouse and other smokers in the household had smoked during the subject's adulthood, but much lower for the number of hours per day of exposure during adulthood (table 5). Because of the small number of subjects living with their parents after age 18 years, we did not calculate correlation coefficients for these variables.

Urine specimens were obtained from 98 per cent of the 149 subjects at the first interview and 95 per cent at the second interview. The median urinary cotinine lev-

els were zero at both interviews, with mean levels of 9.2 ng/mg of creatinine at the first interview and 7.3 ng/mg of creatinine at the second interview. Cotinine levels varied widely with the total number of smokers and the total number of hours of exposure to tobacco smoke (in various situations) during the 24 hours prior to urine collection at both the first interview (figures 1 and 2) and the second interview (data not shown). The cotinine levels correlated only modestly with the questionnaire measures of exposure (table 6).

We also assessed the stability of data on exposure, as measured by questionnaire and by cotinine level (table 6). At the first

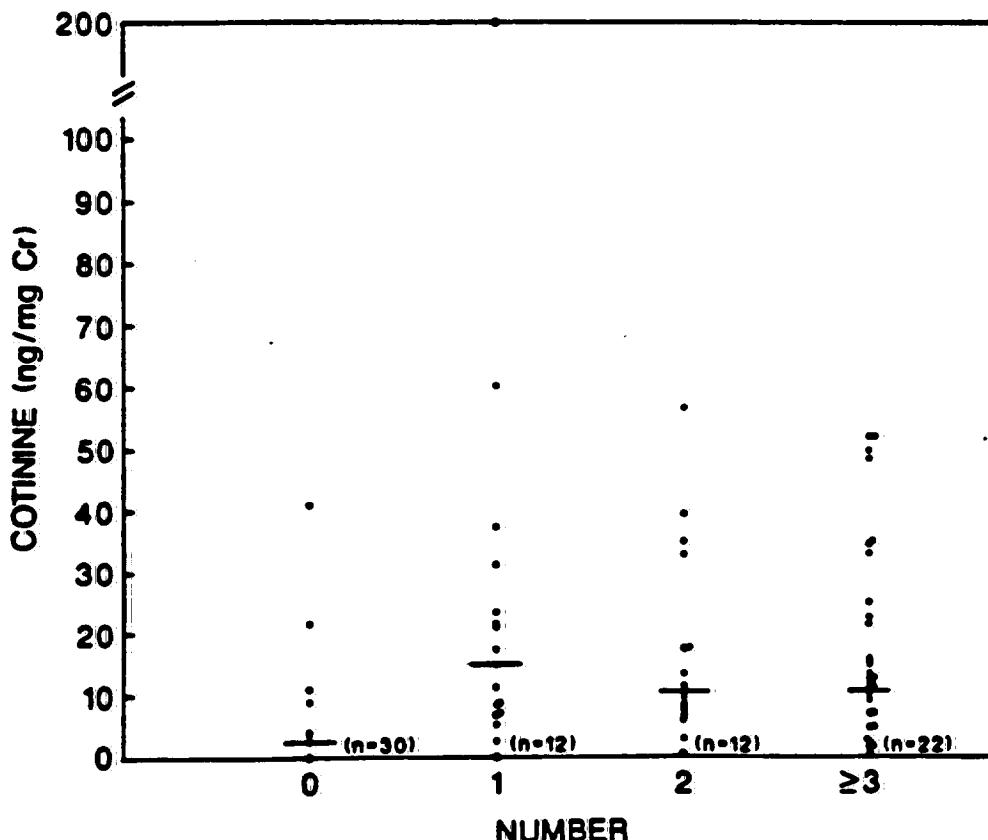


FIGURE 1. Urinary cotinine levels, standardized to urinary creatinine (Cr) concentration, among nonsmokers interviewed about tobacco smoke exposure, by the total number of cigarette smokers the subject reported being exposed to during the 24 hours prior to the first interview. Bars show the mean cotinine level for each group. Values in parentheses indicate the number of subjects with nondetectable levels of cotinine. New Mexico, 1988.

202338C535

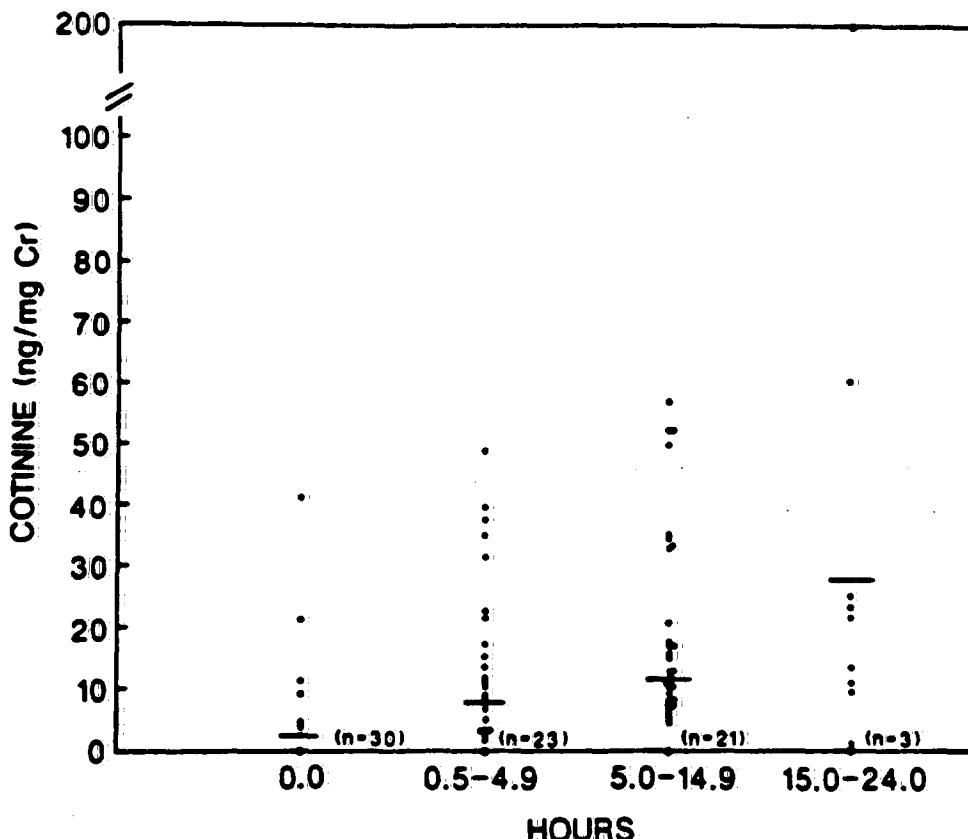


FIGURE 2. Urinary cotinine levels, standardized to urinary creatinine (Cr) concentration, among nonsmokers interviewed about tobacco smoke exposure, by the self-reported total number of hours that the subject was exposed to cigarette smoke during the 24 hours prior to the first interview. Bars above the mean cotinine level for each group. Values in parentheses indicate the number of subjects with nondetectable levels of cotinine. New Mexico, 1986.

and second interviews, the mean responses for the reported number of cigarette smokers that the subjects had been exposed to during the previous 24 hours were 2.1 and 1.8, respectively, with 20 subjects at the first interview and 22 subjects at the second interview reporting exposures in "crowds." For the total number of hours of exposure during the previous 24 hours, the mean responses at the first and second interviews were 5.1 and 4.6, respectively. Both the questionnaire variables and the cotinine data indicated a relatively stable pattern of exposure. The Spearman correlation coefficients were somewhat higher for the

questionnaire-based indexes than for urinary cotinine levels.

#### DISCUSSION

In a group of adult nonsmokers, we found high reliability for reports on parental smoking and on smoking by others in the home (table 2) but lower reliability for semiquantitative exposure measures (tables 3-5). Mean levels of urinary cotinine increased with exposure to cigarette smoke compared with no exposure ( $n = 37$ ) (figures 1 and 2). However, within specific levels of exposure, the cotinine levels varied widely. Across the follow-up period of sev-

2023380536

TABLE 6  
*Spearman correlations between measures of exposure to environmental tobacco smoke during the 24 hours prior to interview, New Mexico, 1986*

Exposure variable	No.	<i>r</i>
Total no. of smokers to which subject was exposed:		
Responses at the first and second interviews	143	0.50
Response at the first interview and cotinine level	143	0.24
Response at the second interview and cotinine level	139	0.21
Total no. of hours that subject was exposed to cigarette smoke		
Responses at the first and second interviews	144	0.62
Response at the first interview and cotinine level	145	0.32
Response at the second interview and cotinine level	138	0.29
Cotinine level:		
Levels at the first and second interviews	140	0.45

eral months, exposures to environmental tobacco smoke were relatively stable, as were urinary cotinine levels (table 6). Most subjects were able to provide responses to the questions on maternal smoking during pregnancy, parental smoking during childhood, and smoking by a spouse during adulthood (tables 2 and 3).

Several limitations of these data must be considered. Because a standard for validating a lifetime history of exposure to environmental tobacco smoke is unavailable, we used repeatability as an index of the quality of questionnaire responses. We addressed the reliability of questions on lifetime exposure at home, but not in the workplace, an important source of exposure for a substantial proportion of adults (11). Interview with a volunteer subject does not replicate the usual setting of a case-control study, the design most often used to examine lung cancer and passive smoking (1). In that setting, recall bias by ill subjects may affect reliability of questionnaire responses in comparison with a volunteer population.

Similar observations on the reliability of questionnaire data on passive smoking were recently reported by Pron et al. (12). These investigators interviewed 117 subjects, controls in a case-control study of lung cancer, on two occasions separated by an average of six months. Smoking by spouses was reported with high reliability ( $\kappa = 0.89$  for both wife and husband). Repeatability was somewhat lower for smoking by the mother ( $\kappa = 0.76$ ) and by the father ( $\kappa = 0.44$ ). As in the present study, repeatability of quantitative estimates of duration of exposure was lower than for the categorical descriptions of smoking by household members.

Although neither the investigation of Pron et al. (12) nor the present study directly addresses validity of questionnaires on lifetime passive smoking, the validity of subjects' reports on smoking by parents and spouses has been described. Sandler and Shore (13) compared responses on parents' smoking given by cases and controls with responses given by the parents or siblings of the index cases. Concordance was high for whether the parents had ever smoked, although the agreement was somewhat better for smoking by the mother than for smoking by the father. Responses concerning numbers of cigarettes smoked did not agree as highly. In a follow-up study of a nationwide sample, children's responses on smoking by their deceased parents closely agreed with the information given 10 years previously by the parents themselves (14). Other studies have shown that people generally report the smoking habits of their spouses correctly (14-19). However, people's reporting of quantitative aspects of the smoking behavior of their spouses tends to be less valid (16, 18, 19).

Smoking by parents during childhood and by a spouse during adulthood represent the most important sources of household exposure to environmental tobacco smoke. The studies of subject reports for parents and spouses indicate good validity of responses on smoking by these household

2023380537

members; the study of Pron et al. (12) and the present study show that these reports are also highly reliable. Thus, exposure measures based on cigarette smoking status of parents and of spouses, as reported by an index subject, are reported with a high degree of validity and reliability, although these measures may only crudely quantitate the dose of biologically relevant tobacco smoke components. In contrast, the accuracy of more quantitative measures of smoking by these household members is lower. The resulting misclassification may explain the failure to find exposure-response relations for passive smoking and lung cancer in some studies (1, 20).

We also compared responses to questions on exposure during the previous 24 hours with urinary cotinine level. The time period for the questionnaire was limited to the previous 24 hours to reduce bias from faulty recall. However, since this period is approximately the half-life of cotinine in nonsmokers (21, 22), the cotinine level represents not only exposure during the 24 hours covered by the questionnaire but prior exposure as well.

We found modest correlations between the questionnaire-based measures of exposure and urinary cotinine levels (table 6). The level of correlation must be interpreted in the context of the different lengths of time of exposure assessed by the questionnaire and by the urinary cotinine level. Furthermore, at a given level of nicotine exposure, urinary cotinine level is also influenced by uptake, metabolism, and excretion, which are likely to vary among individuals.

Coultas et al. (23) found that questionnaire measures of household exposure were not strong predictors of salivary cotinine level. In 247 adult nonsmokers with a detectable cotinine level, the subject's age, the number of cigarettes smoked per day by the spouse, and the number of cigarettes smoked per day by other smokers in the household explained only 2 per cent of the variance in cotinine levels for females and 16 per cent of the variance for males. Even

in active smokers, questionnaire responses on smoking behavior do not tightly predict cotinine concentrations in body fluids (24-27). Higher correlations between urinary cotinine levels and reported exposure to cigarette smoke have been reported for young children (28). The higher correlations in the studies of young children probably reflect the time-activity patterns in this age group (29); parental smoking in the household is generally the dominant source of exposure.

In adults, the weak relation between cotinine level and reported smoke exposure implies that a single cotinine measurement should not be used to estimate exposure for individuals (23). However, in our subjects, cotinine levels varied among exposure groups (figures 1 and 2), suggesting that cotinine measurements might be used as an index of mean exposure for members of a particular exposure group.

Nonsmokers are exposed to environmental tobacco smoke in many different environments, including the home, the workplace, and other private and public locations. Since subjects in an epidemiologic investigation cannot be expected to comprehensively describe the extent of exposure in each of these environments, misclassification of the amount of exposure to environmental tobacco smoke must be anticipated from the use of questionnaires. However, subjects can provide valid and reliable reports concerning the smoking status of household members. The combination of questionnaires and biologic markers offers a feasible approach for assessing recent exposure to environmental tobacco smoke.

#### REFERENCES

1. US Department of Health and Human Services. *The health consequences of smoking: a report of the Surgeon General*. Rockville, MD: US Department of Health and Human Services, 1986. (DHHS [CDC] publication no. 87-8398).
2. National Research Council. *Environmental tobacco smoke: measuring exposure and assessing health effects*. Washington, DC: National Academy Press, 1986. (ISBN 0-309-03730-1).
3. Centers for Disease Control. *Cigarette smoking in*

2023380538

- the United States. 1986. MMWR 1987;36:581-5.
4. Samet JM, Coulas DB, Howard CA, et al. Respiratory diseases and cigarette smoking in an Hispanic population in New Mexico. *Am Rev Respir Dis* 1988;137:815-19.
  5. Langone JJ, Gjika HB, Van Vunakis V. Nicotine and its metabolites: radioimmunoassays for nicotine and cotinine. *Biochemistry* 1973;12:5025-30.
  6. Faulkner WR, King JW. Renal function. In: Tietz NW, ed. *Fundamentals of clinical chemistry*. Philadelphia, PA: WB Saunders Co, 1976:975-1014.
  7. Fleiss JL. Measuring nominal scale agreement among many raters. *Psychol Bull* 1971;76:378-82.
  8. MacPherson M, Willett WC. Misinterpretation and misuse of the Kappa statistic. *Am J Epidemiol* 1987;126:161-9.
  9. Colton T. *Statistics in medicine*. Boston, MA: Little, Brown and Company, 1974.
  10. SAS Institute, Inc. *SAS user's guide: statistics*. Version 5 edition, 1985. Cary, NC: SAS Institute, Inc, 1985.
  11. Friedman GD, Petitti DB, Bewoi RD. Prevalence and correlates of passive smoking. *Am J Public Health* 1983;73:401-5.
  12. Pron GE, Burch JD, Howe GR, et al. The reliability of passive smoking histories reported in a case-control study of lung cancer. *Am J Epidemiol* 1988;127:267-73.
  13. Sandler DP, Shore DL. Quality of data on parents' smoking and drinking provided by adult offspring. *Am J Epidemiol* 1986;124:768-78.
  14. McLaughlin JK, Dietz MS, Mehl ES, et al. Reliability of surrogate information on cigarette smoking by type of informant. *Am J Epidemiol* 1987;126:144-6.
  15. Rogot E, Reid D. The validity of data from next-of-kin in studies of mortality among immigrants. *Int J Epidemiol* 1975;4:51-4.
  16. Kolonel LN, Hirohata T, Nomura AMY. Adequacy of survey data collected from substitute respondents. *Am J Epidemiol* 1977;106:476-84.
  17. Pershagen G. Validity of questionnaire data on smoking and other exposures with special reference to environmental tobacco smoke. *Eur J Respir Dis* 1984;133(suppl):76-80.
  18. Humble CG, Samet JM, Skipper BE. Comparison of self- and surrogate-reported dietary information. *Am J Epidemiol* 1984;119:85-96.
  19. Lorchon ML, Samet JM. An assessment of the validity of questionnaire responses provided by a surviving spouse. *Am J Epidemiol* 1986;123:481-9.
  20. Humble CG, Samet JM, Pathak DR. Marriage to a smoker and lung cancer risk. *Am J Public Health* 1987;77:598-602.
  21. Kyerematen GA, Damiano MD, Dvorchik BH, et al. Smoking-induced changes in nicotine disposition: application of a new HPLC assay for nicotine and its metabolites. *Clin Pharmacol Ther* 1982;32:769-80.
  22. Sepkovic DW, Haley NJ, Hoffmann D. Elimination from the body of tobacco smoke products by smokers and passive smokers. (Letter). *JAMA* 1986;256:863.
  23. Coulas DB, Howard CA, Peake GT, et al. Salivary cotinine levels and involuntary tobacco smoke exposure in children and adults in New Mexico. *Am Rev Respir Dis* 1987;136:306-9.
  24. Benowitz NL, Hall SM, Herning RI, et al. Smokers of low-yield cigarettes do not consume less nicotine. *N Engl J Med* 1983;309:139-42.
  25. Abrams DB, Follick MJ, Biener L, et al. Saliva cotinine as a measure of smoking status in field settings. *Am J Public Health* 1987;77:845-8.
  26. Lee PN. Lung cancer and passive smoking: association an artefact due to misclassification of smoking habits? *Toxicol Lett* 1987;35:157-62.
  27. Pierce JP, Dwyer T, DiGuusto E, et al. Cotinine validation of self-reported smoking in commercially run community surveys. *J Chronic Dis* 1987;40:689-95.
  28. Greenberg RA, Haley NJ, Etzel RA, et al. Measuring the exposure of infants to tobacco smoke. *N Engl J Med* 1984;310:1075-8.
  29. Harlow DP, Marbury M, Samet J, et al. Relating indoor NO<sub>2</sub> levels to infant personal exposures. *Atmosph Environ* 1987;21:369-76.

2023386539

# Variability of Measures of Exposure to Environmental Tobacco Smoke in the Home<sup>1-4</sup>

DAVID B. COULTAS,<sup>1</sup> JONATHAN M. SAMET, JOHN F. MCCARTHY, and JOHN D. SPENGLER

## Introduction

Numerous epidemiologic investigations have examined the adverse effects of passive smoking on children and adults; the evidence is sufficiently compelling to establish passive smoking as a cause of disease in nonsmokers (1, 2). Both the 1986 Surgeon General's Report (1) and the National Research Council (2) have concluded that passive smoking causes increased lower respiratory illness in infants, increased respiratory symptoms in children, reduced lung growth during childhood, and lung cancer in nonsmokers. Although health effects of passive smoking have been convincingly demonstrated, additional research is needed to address unresolved issues concerning this preventable exposure. For example, more precise description of exposure-response relations should be achieved for the already established health effects. Uncertainties concerning the adverse effects of passive smoking in the workplace and on the occurrence of ischemic heart disease must also be resolved.

The conduct of this research would be facilitated by improved methods for exposure assessment. In most epidemiologic studies on passive smoking published to date, exposure to environmental tobacco smoke, the combination of exhaled mainstream smoke and sidestream smoke, has been assessed by questionnaire. However, exposure to environmental tobacco smoke can also be estimated with air monitoring and measurement of biologic markers in body fluids, such as salivary cotinine. Biologic markers are increasingly emphasized as a standard for validating questionnaire responses. To characterize the relationships among these alternative approaches for assessing passive smoking in the home environment, we conducted a prospective study of 10 households. We periodically collected questionnaire information on exposure and measured respirable particles and nicotine in air samples and urinary and salivary cotinine in the 20 nonsmokers in these households.

**SUMMARY** We assessed the variability of four markers of environmental tobacco smoke exposure in 10 homes with 20 nonsmoking and 11 smoking household members. We obtained exposure questionnaires, saliva and urine for cotinine, and air particle samples for respirable particles and nicotine on 10 sampling days: every other day over 10 days, and then 1 day every other week over 10 wk. The mean concentrations of respirable particles in the 10 homes ranged from 22.4 to 76.8  $\mu\text{gm}^{-3}$ , and concentrations of nicotine ranged from 0.0 to 0.9  $\mu\text{g}/\text{m}^3$ . Linear regression models that included indicator variables for self-reported exposure explained 9 and 6% of the variability of the respirable particle and the nicotine concentrations, respectively. The individual mean urinary cotinine levels standardized to creatinine concentration ranged from 3.0 to 84.8  $\text{ng}/\text{mg Cr}$ , and for salivary cotinine the mean levels ranged from 0.5 to 4.3  $\text{ng}/\text{ml}$ . Indicator variables for self-reported exposure explained 8 and 23% of the variability of the urinary and salivary cotinine levels, respectively. We conclude that because of the marked variability of these measures, multiple measurements are needed to establish a stable profile of exposure to environmental tobacco smoke in the home.

AM REV RESPIR DIS 1987; 135: 482-488

## Methods

### Sample Selection

Between February and December 1986, 149 nonsmoking volunteers, 18 years of age and older, were recruited from Albuquerque and surrounding communities to participate in a study of the accuracy of questionnaire assessment of exposure to environmental tobacco smoke (3). From this sample, we selected 10 subjects living with at least one cigarette smoker and requested the participation of the entire household for this investigation. The households were selected on the basis of willingness to participate and location, and were not intended to be representative of the original sample.

### Data Collection

Between March and October 1986, we obtained exposure questionnaires, saliva and urine, and air particle samples on 10 sampling days: every other day over 10 days, and then 1 day every other week over 10 wk. The questionnaires and saliva and urine specimens were obtained at the end of a 24-h air monitoring period (described below). From the questionnaires, we determined the reported number of smokers and number of hours that the subjects were exposed during the previous 24 h to cigarettes, cigars, and pipes at home, at work or school, in a vehicle, and in other places. Questionnaires were self-completed by the adults, and by a parent for children 14 years of age and younger. Spot saliva and urine specimens were obtained and frozen at -20° C until the cotinine assays were performed.

### Cotinine Assay

Cotinine was quantitated by a double antibody radioimmunoassay, as described by Langone and coworkers (4). A specific antiserum produced in rabbits was supplied by Dr. Helen Van Vunakis (Brandeis University). Urine samples were diluted 1:4 for the assay. The sensitivity of the assay in our hands was 36 pg/tube or 0.78 ng/ml of urine (4.204 pmol/L). Urine creatinine concentrations were determined by the Jaffe reaction (5), and the cotinine concentrations were standardized to the creatinine concentrations. Assays were performed without knowledge of questionnaire responses.

(Received in original form September 26, 1989 and in revised form February 3, 1990)

<sup>1</sup> From the New Mexico Tumor Registry, Cancer Center, University of New Mexico Medical Center, Albuquerque, New Mexico, and the Department of Environmental Science Engineering, Harvard School of Public Health, Boston, Massachusetts.

<sup>2</sup> Supported by Grant No. EPA CR811630 from the Environmental Protection Agency.

<sup>3</sup> Presented in part at the Air Pollution Control Association Specialty Conference on Combustion Processes and the Quality of the Indoor Environment, Niagara Falls, New York, September 1984.

<sup>4</sup> Correspondence and requests for reprints should be addressed to David B. Coultas, M.D., New Mexico Tumor Registry, 900 Camino de Salud NE, Albuquerque, NM 87131.

<sup>5</sup> Recipient of an Edward Livingston Trudeau Scholar Award from the Americas Lung Association.

2023380540

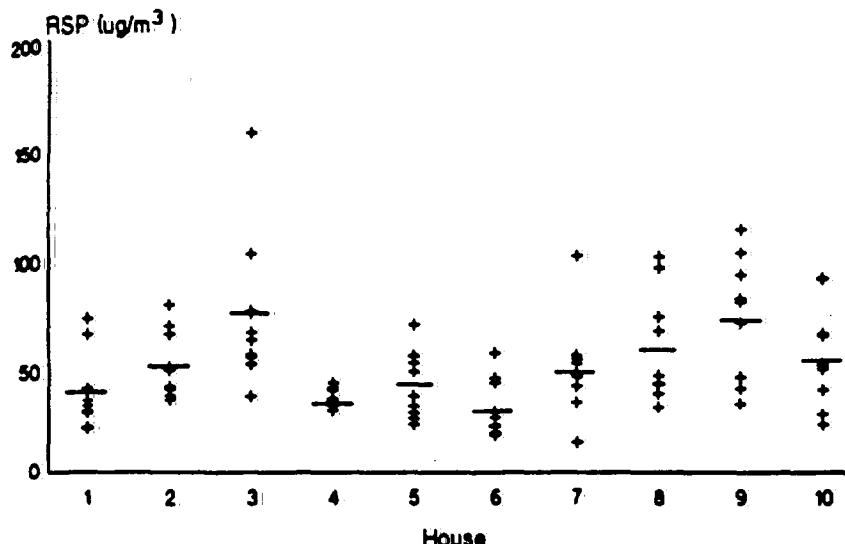


Fig. 1. Respirable particle concentrations (RSP) measured during 24-h sampling periods in 10 homes with at least one cigarette smoker. The bars indicate the mean levels for each home.

#### Particle Measurements

In the major activity room of each home, Harvard School of Public Health impactors (6), operating at a flow rate of 4 L/min, were used to collect respirable particles and gaseous nicotine samples. Through a timed solenoid switching valve, two impactors used a common, mass-flow controlled pump, and each impactor operated on alternate 15-min collection cycles. Respirable particle samples, 2.5  $\mu\text{m}$  in diameter or less, were collected on Teflon<sup>®</sup> filters (Membrana, Inc., Pleasanton, CA), and nicotine was collected on sodium-bisulfate-treated glass fiber filters (Millipore Corp., Bedford, MA) to minimize its volatilization. After extraction from the filter, analysis for nicotine was done on a Shimadzu GC7A gas chromatograph (Columbia, MD) with a flame ionization detector. The nicotine collection and extraction procedure is a modification of that described by Hammond and coworkers (7). The recovery of nicotine by this procedure has been shown to be 98% efficient. The sensitivity for detection of respirable particles and nicotine was 5.0  $\mu\text{g}$  and 0.05 ppm, respectively.

#### Data Analysis

Variability of questionnaire responses, respirable particle and nicotine concentrations, and urinary cotinine levels were assessed with univariate analyses. From the questionnaire responses, we used the total number of household smokers, including cigarette, cigar, and pipe smokers, and the total number of hours exposed as the measures of home exposure. The predominant source of tobacco smoke was from cigarette smoking. During the entire sampling period, there were only 4 days in which any subject reported exposure to a cigar smoker, and none reported exposure to a pipe smoker.

Data analyses were performed with standard programs of the Statistical Analysis System (8).

#### Results

The 10 households included 11 cigarette smokers and 20 nonsmokers, 11 females and nine males 1.5 to 74 yr of age. The homes included eight unattached single family houses, one mobile home, and one apartment.

Reports on exposure to tobacco smoke in the home were obtained for all 10 sampling days from 17 subjects, and for 9 days from three subjects. The reported number of cigarette smokers in the home per day did not vary widely. The median number (range) of smokers per day was one for 18 of the nonsmoking subjects (zero to 10), zero for one subject (zero to 1), and four for one subject (2 to 25). Greater variability was reported for the number of hours exposed to cigarette smoke in the home, with the median number of hours ranging from zero to 11 h.

Respirable particle and nicotine concentrations were obtained for 99% of the sampling days (figures 1 and 2). The mean concentrations of respirable particles in the 10 homes ranged from 32.4  $\mu\text{g}/\text{m}^3$  (SD = 13.1) to 76.9  $\mu\text{g}/\text{m}^3$  (SD = 32.9), and concentrations of nicotine ranged from 0.6  $\mu\text{g}/\text{m}^3$  (SD = 0.69) to 6.9  $\mu\text{g}/\text{m}^3$  (SD = 8.2). Spearman's correlation coefficient between the respirable particle concentrations and the nicotine concentrations was 0.54 ( $n = 99$ ,  $p = 0.0001$ ).

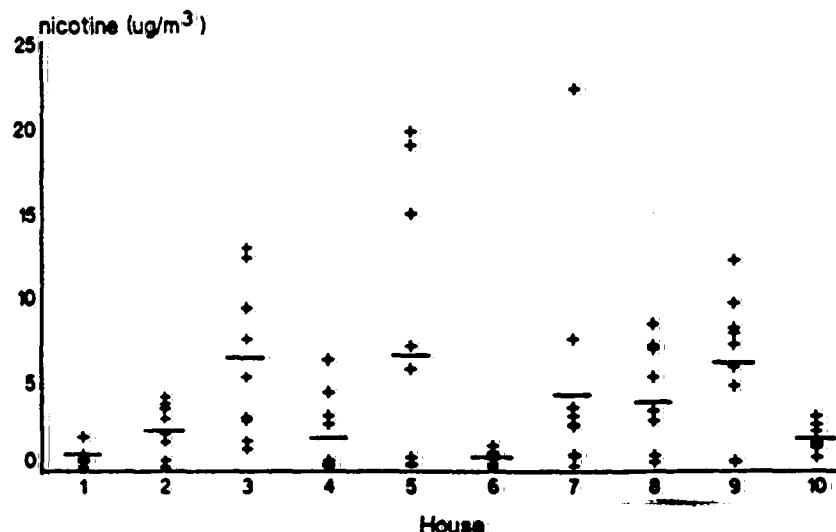


Fig. 2. Atmospheric nicotine concentrations measured during 24-h sampling periods in 10 homes with at least one cigarette smoker. The bars indicate the mean levels for each home. Levels of nicotine were undetectable on 1 or more days in Houses 1 ( $n = 3$ ), 2 ( $n = 2$ ), 4 ( $n = 2$ ), and 6 ( $n = 1$ ).

2023380541

TABLE 1  
COEFFICIENTS OF DETERMINATION FOR THREE LINEAR REGRESSION MODELS\* PREDICTING RESPIRABLE PARTICLE AND NICOTINE CONCENTRATIONS IN AIR SAMPLES FROM 10 HOMES, NEW MEXICO, 1986

Dependent Variable	R <sup>2</sup>		
	Model 1	Model 2	Model 3
Respirable particles, $\mu\text{g}/\text{m}^3$	0.34	0.08	0.09
Nicotine, $\mu\text{g}/\text{m}^3$	0.28	0.04	0.06

\* Independent variables: Model 1 = HOUSE (1 to 10, representing the 10 homes); Model 2 = NUMBER (zero versus ≥ 1 smokers); + SEASON (March–April versus May–October). Model 3 = NUMBER (zero versus ≥ 1 smokers) + HOURS (continuous) + SEASON (March–April versus May–October).

TABLE 2  
REGRESSION COEFFICIENTS FOR MODEL THREE\* PREDICTING RESPIRABLE PARTICLE AND NICOTINE CONCENTRATIONS IN AIR SUPPLY FROM 10 HOMES, NEW MEXICO, 1986

Regression Coefficients for Model 3			
One or More Smokers	HOURS	Cold Months	
Respirable particles, $\mu\text{g}/\text{m}^3$	+17.3 (-3.0, 37.7) <sup>†</sup>	+0.4 (-1.0, 1.8)	+8.9 (-1.1, 18.8)
Nicotine, $\mu\text{g}/\text{m}^3$	+2.1 (-2.7, 5.9)	+0.2 (-0.1, 0.5)	-0.7 (-2.5, 1.1)

\* See text and table 1 for description of Model 3.

<sup>†</sup> 95% confidence intervals shown in parentheses.

The variability of respirable particle and nicotine concentrations for the two sampling periods, every other day or every other week, were described with one-way analysis of variance. For the respirable particle concentrations, the intra-house mean square error, describing the extent of variation for a particular house-

hold, was greatest for sampling every other day (516.8) compared with every other week (238.7). A contrasting pattern of variation was observed for nicotine, with mean square errors of 3.6 and 19.0 for every other day and every other week, respectively.

For the particle and nicotine measure-

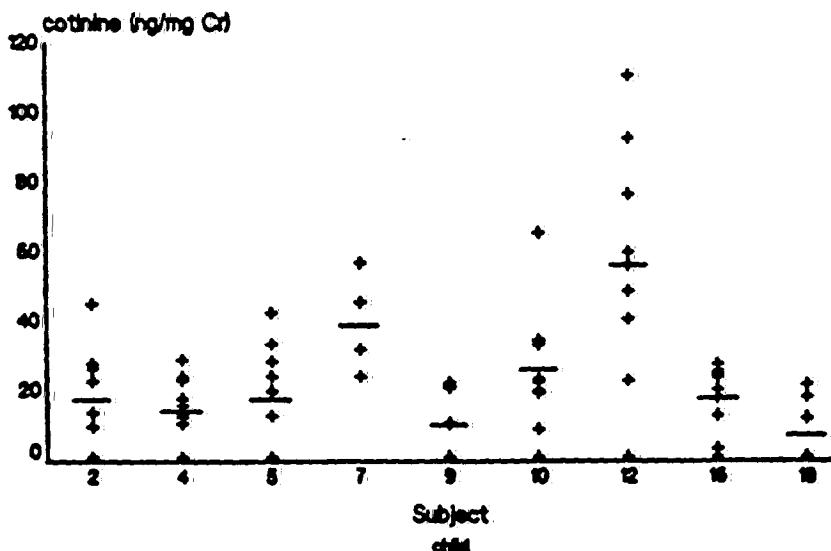


Fig. 3. Urinary cotinine concentrations standardized to urinary creatinine concentration in nine nonsmoking children from homes with at least one cigarette smoker. The bars indicate the mean levels for each child. Levels of urinary cotinine were undetectable on 1 or more days for Subjects 2 ( $n = 2$ ), 4 ( $n = 2$ ), 5 ( $n = 3$ ), 9 ( $n = 4$ ), 10 ( $n = 1$ ), 12 ( $n = 1$ ), 16 ( $n = 1$ ), and 18 ( $n = 5$ ).

ments, we used linear regression to examine factors influencing the concentrations and the variability of the concentrations. A model that included variables representing each of the 10 houses explained the greatest amount of variability, as shown by the magnitude of the  $R^2$  value (table 1). Compared with the model with the variables for individual homes, the models that included number of smokers explained markedly lower percentages of the variability of levels of nicotine and particles. Although not statistically significant, increases in respirable particles were associated with exposure to one or more cigarette smokers in the home and with the colder months, March and April (table 2). There was no association of particle levels with the number of hours of exposure. Nicotine levels increased, although not significantly, with exposure to smokers in the home, but were not predicted by the season (table 2).

Cotinine levels were obtained on 187 urine specimens from 20 nonsmokers, and 153 saliva specimens were obtained from 16 nonsmokers. We were unable to obtain saliva specimens from four children, all 4 yr of age or younger. The individual mean urinary cotinine levels standardized to urinary creatinine concentration ranged from 3.9 ng/mg Cr ( $SD = 6.5$ ) to 55.8 ng/mg Cr ( $SD = 32.0$ ). For salivary cotinine, the mean levels ranged from 0.9 ng/ml ( $SD = 0.8$ ) to 4.3 ng/ml ( $SD = 1.4$ ). The mean urinary cotinine levels and variability tended to be greater in the children than in the adults (figures 3 and 4) (data not shown for salivary cotinine). Spearman's correlation between the urinary cotinine and salivary cotinine concentrations was 0.32 ( $n = 153$ ,  $p = 0.0001$ ). Correlations between the cotinine levels and the atmospheric markers were highest for salivary cotinine and nicotine (table 3).

As for the atmospheric markers, we

TABLE 3  
SPEARMAN'S CORRELATION COEFFICIENTS BETWEEN COTININE LEVELS IN URINE AND SALIVA AND RESPIRABLE PARTICLES AND NICOTINE, NEW MEXICO, 1986

Urinary cotinine, $n = 187$
Respirable particles
Nicotine
Salivary cotinine, $n = 153$
Respirable particles
Nicotine

2023380542

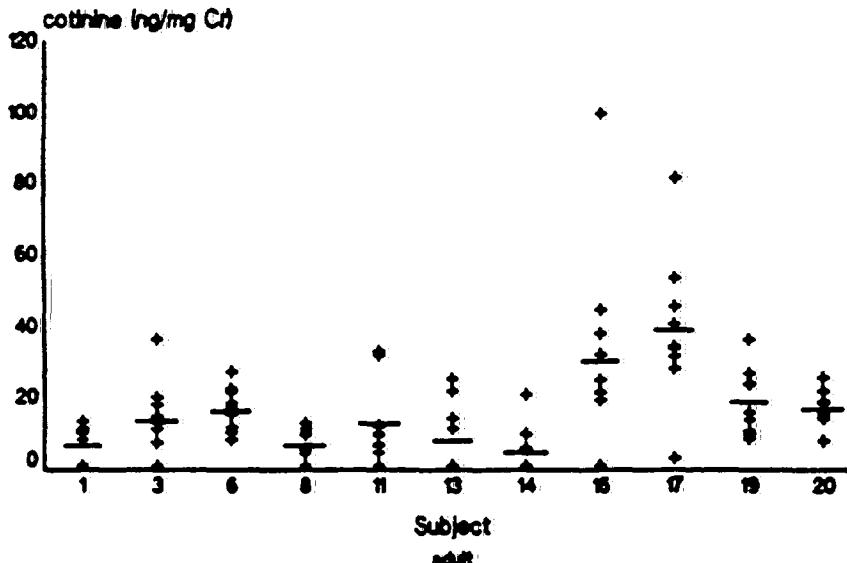


Fig. 4. Urinary cotinine concentrations standardized to urinary creatinine concentration in 20 nonsmoking adults from homes with at least one cigarette smoker. The bars indicate the mean levels for each adult. Levels of urinary cotinine were undetectable on 1 or more days for Subjects 1 ( $n = 4$ ), 3 ( $n = 2$ ), 8 ( $n = 2$ ), 11 ( $n = 1$ ), 13 ( $n = 6$ ), 14 ( $n = 6$ ), and 15 ( $n = 2$ ).

TABLE 4  
COEFFICIENTS OF DETERMINATION FOR THREE LINEAR REGRESSION MODELS' PREDICTING URINARY AND SALIVARY COTININE CONCENTRATIONS IN NONSMOKERS EXPOSED TO TOBACCO SMOKE, NEW MEXICO, 1986

Dependent Variable	R <sup>2</sup>		
	Model 1	Model 2	Model 3
Urinary cotinine, ng/mg Cr	0.47	0.08	0.08
Salivary cotinine, ng/ml	0.57	0.08	0.23

\* Independent variables: Model 1 = INDIVIDUAL (1 to 20, representing the 20 nonsmoking individuals); Model 2 = NUMBER (zero versus > 1 smokers) + SEASON (March-April versus May-October); + AGE GROUP (< 18 versus > 18 yr); Model 3 = NUMBER (zero versus > 1 smokers) + HOURS (continuous) + SEASON (March-April versus May-October) + AGE GROUP (< 18 versus > 18 yr).

used one-way analysis of variance to describe the variability in urinary and salivary cotinine concentrations during the two sampling periods: every other day or every other week. In contrast to the atmospheric markers, the variability in cotinine levels was comparable for the two periods. The intraindividual mean

square errors for urinary cotinine were 175.8 and 194.8, and for salivary cotinine it was 0.9 and 0.7.

For the urinary and salivary cotinine levels, we also examined determinants of variability and concentration with linear regression. Models that included indicator variables for the 20 nonsmoking

subjects explained 47 and 57% of the variability in cotinine levels, respectively (table 4). Compared with this model, other models that included exposure to environmental tobacco smoke and age group explained much lower proportions of the variability. Urinary cotinine levels were significantly ( $p < 0.05$ ) higher among children than among adults (table 5). Although the effect was not significant, exposure to one or more smokers resulted in higher urinary cotinine levels than did no exposure. The number of hours of reported exposure and the season were not significant predictors of cotinine level. For salivary cotinine level, the hours of exposure was the only significant predictor.

Prediction of level of urinary or salivary cotinine was not greatly improved with the use of respirable particles or nicotine as independent variables. The proportions of the variability in the urinary cotinine levels explained by respirable particle and nicotine concentrations were 0.03 and 0.04, respectively. For salivary cotinine, the corresponding  $R^2$  values were only slightly higher at 0.07 and 0.13, respectively.

#### Discussion

Environmental tobacco smoke is a complex mixture of gases and particles that changes as it ages. Personal exposure to environmental tobacco smoke is determined by the nonsmoker's activity pattern; exposure may be received in the diverse microenvironments encountered throughout the course of day-to-day activities. For many nonsmokers, the home is a predominant location of exposure (9). In this investigation, we assessed methods for measuring exposure to environmental tobacco smoke in the home that can be used for epidemiologic research: air monitoring, questionnaires, and biologic markers.

In other populations, cigarette smok-

TABLE 5  
REGRESSION COEFFICIENTS FOR MODEL THREE\* PREDICTING URINARY AND SALIVARY COTININE CONCENTRATIONS IN NONSMOKERS EXPOSED TO TOBACCO SMOKE, NEW MEXICO, 1986

	Regression Coefficients* Model 3			
	One or More Smokers	HOURS	SEASON	AGE GROUP
Urinary cotinine, ng/mg Cr	+3.4 (-4.8, 11.6)†	+0.8 (0.0, 1.8)	-0.2 (-5.8, 5.4)	+0.6 (0.0, 10.8)
Salivary cotinine, ng/ml	+0.13 (-0.08, 0.36)	+0.15 (0.08, 0.21)	-0.02 (-0.41, 0.37)	-0.81 (-1.34, -0.28)

\* See text and table 4 for description of Model 3.

† 95% confidence intervals shown in parentheses.

2023380543

ing has been shown to be a strong source of respirable particles in the home (1, 10, 11). Spengler and coworkers (10) estimated that the average increase in the indoor concentration of respirable particles was  $20 \mu\text{g}/\text{m}^3$  for each smoker. We estimated an average increase of  $17 \mu\text{g}/\text{m}^3$  for one or more smokers (table 2); the average concentrations in the New Mexico homes (figure 1) were above the mean of  $24 \mu\text{g}/\text{m}^3$  in nonsmoking homes from six U.S. cities (10). Nicotine was present on most sampling days (figure 2). The moderate correlation between the nicotine and respirable particle concentrations (Spearman's  $r = 0.54$ ) confirms the importance of tobacco smoking as a source of particulate pollution in the home. Little data have been reported on nicotine concentrations in the home (1, 2); the levels in the New Mexico homes were somewhat lower than an average concentration of  $11.2 \mu\text{g}/\text{m}^3$  reported by Muramatsu and coworkers (12) for three homes in Japan. However, the results from our investigation and the Japanese study are not directly comparable because the Japanese data were from personal samples. Furthermore, information on intensity or duration of exposure to tobacco smoke was not provided for the Japanese homes. In a recent study in North Carolina, the homes of 27 children were monitored overnight for nicotine with a sampler that was located near the child (13). The average nicotine concentration in homes with smokers was  $3.74 \mu\text{g}/\text{m}^3$ , with a range from about 1 to  $7 \mu\text{g}/\text{m}^3$ . The higher levels in our study may reflect the differing sampling strategies; the nicotine sampler remained in the activity room throughout the monitoring period in our study, but it was moved to the child's bedroom in the North Carolina study when the child slept.

Questionnaires on exposure to environmental tobacco smoke generally assess the strength of the source, e.g., the number of smokers or the number of cigarettes consumed, and the duration of exposure. The concentration of environmental tobacco smoke, however, depends not only on the source strength but on room size, mixing, adsorption of smoke components, and the rate of exchange of indoor with outdoor air. Personal exposure also varies with the nonsmoker's proximity to the smoker. Questionnaires cannot comprehensively and accurately assess each of these factors.

Not surprisingly, we found that the questionnaire responses were poor predictors of concentrations of respirable

particles and nicotine (table 1). The highest  $R^2$  values were obtained with a regression model that included variables for the individual homes; presumably, these variables represented characteristics of the homes, many of them unmeasurable, that determined concentrations at a given level of smoking.

Cotinine, nicotine's major metabolite, has a half-life of 20 to 40 h in nonsmokers (1). It can serve as a specific biologic marker of exposure to environmental tobacco smoke that has been received over a period of days. At any given level of nicotine exposure, cotinine levels in body fluids are also determined by uptake, metabolism, and excretion (1). In regression analyses to predict cotinine concentrations, the models that included variables for the individual subjects gave the highest  $R^2$  values (table 4). Models including only the questionnaire-derived exposure measures or the atmospheric markers had low  $R^2$  values. Our findings in a large population-based survey were similar (14). In 247 nonsmoking adults with a detectable cotinine level, variables for subject age, number of cigarettes smoked by the spouse, and number of cigarettes smoked by other household smokers explained only 2% of the variance of salivary cotinine level for females, and 16% of the variance for males.

In epidemiologic investigations of the adverse health effects of environmental tobacco smoke, questionnaires have been the sole approach for assessing exposure (1, 2). Air monitoring and biologic markers represent promising and feasible approaches for assessing exposure to environmental tobacco smoke. For the home environment, our data demonstrate that indexes of exposure to environmental tobacco smoke based on questionnaires, biologic markers, and air monitoring are not tightly correlated. At a particular level of exposure, as assessed by inventory of household smokers, concentrations of respirable particles and nicotine vary widely, as do levels of salivary and urinary cotinine. The variability of the atmospheric and biologic markers must be considered in using them as standards for assessing misclassification by questionnaires. For environmental tobacco smoke exposure at home, our data suggest that single measurements of either levels of environmental tobacco smoke components or of biologic markers are not adequate for characterizing usual exposure. Multiple measurements are needed. It may be misleading to assess the va-

lidity of questionnaire measures against a single determination of an atmospheric or biologic marker. We suggest that atmospheric and biologic markers offer complementary approaches to questionnaires for assessment of exposure to environmental tobacco smoke, and that these methods should be used together to estimate the magnitude of misclassification from questionnaire responses.

#### Acknowledgment

The writers thank Dr. Helen Van Yunakis for providing the reagents for the radioimmunoassay, and Irene Walkiw for technical assistance in performing the assays.

#### References

- U.S. Department of Health and Human Services. The health consequences of involuntary smoking, a report of the Surgeon General. Rockville, MD: U.S. Department of Health and Human Services, 1986. (DHHS publication no. [CDC] 87-8398).
- National Research Council. Environmental tobacco smoke: Measuring exposure and assessing health effects. Washington, DC: National Academy Press, 1986.
- Coultas DB, Peake GT, Samet JM. Questionnaire assessment of lifetime and recent exposure to environmental tobacco smoke. *Am J Epidemiol* 1989; 130:338-47.
- Lazoglo JJ, Gjika HB, Van Yunakis H. Nicotine and its metabolism. Radioimmunoassays for nicotine and cotinine. *Biochemistry* 1973; 12: 5025-30.
- Faulkner WR, King JW. Renal function. In: Tietz NW, ed. Fundamentals of clinical chemistry. Philadelphia: W. B. Saunders Co, 1976: 975-1014.
- Turner WA, Marple VA, Spengler JD. Indoor aerosol impactor. In: Liu BYH, Pui D, Fries H, eds. Aerosols. New York: Elsevier Science Publishing Co, 1984: 527.
- Hammond SK, Leaderer BP, Roche AC, Schenker M. Collection and analysis of nicotine as a marker for environmental tobacco smoke. *Atmos Environ* 1987; 21:457-62.
- SAS Institute, Inc. SAS user's guide: Statistics, 5th ed. Cary, NC: SAS Institute, Inc., 1985.
- Friedman GD, Pezzati DE, Erwol RD. Prevalence and correlates of passive smoking. *Am J Public Health* 1983; 73:404-5.
- Spengler JD, Dockery DW, Turner WA, Wolfson JM, Ferris BG Jr. Long-term measurements of respirable sulfates and particles inside and outside homes. *Atmos Environ* 1981; 15:23-30.
- Brunekreef B, Boeve JSM. Long-term average suspended particulate concentrations in smokers' homes. *Int Arch Occup Environ Health* 1982; 50:299-302.
- Muramatsu M, Umemoto S, Okada T, Tomita H. Estimation of personal exposure to tobacco smoke with a newly developed nicotine personal monitor. *Environ Res* 1984; 35:218-27.
- Henderson FW, Reid HF, Morris R, et al. Home air nicotine levels and urinary cotinine excretion in preschool children. *Am Rev Respir Dis* 1989; 140:197-201.
- Coultas DB, Howard CA, Peake GT, Skipper BJ, Samet JM. Salivary cotinine levels and involuntary tobacco smoke exposure in children and adults in New Mexico. *Am Rev Respir Dis* 1987; 136:303-A.

2023380549

# A Personal Monitoring Study to Assess Workplace Exposure to Environmental Tobacco Smoke

DAVID B. COULTAS, MD, JONATHAN M. SAMET, MD, JOHN F. McCARTHY, ScD, AND JOHN D. SPENGLER, PhD

**Abstract:** We enrolled 15 nonsmoking volunteers to evaluate the feasibility of measuring personal exposure to environmental tobacco smoke (ETS) at work and to characterize workplace exposures. During one workshift, we obtained questionnaires on exposure, saliva and urine for cotinine, and personal air samples for respirable particles and nicotine. The levels of cotinine, respirable particles, and nicotine varied widely with self-reports of exposure to ETS, but on average increased with increasing exposure. (*Am J Public Health* 1990; 80:988-990.)

## Introduction

While health effects of passive smoking on children and adults have been identified, the principal location of exposure investigated has been the home.<sup>1,2</sup> Workplace exposure has received less attention, and health effects of environmental tobacco smoke (ETS) in the workplace remain controversial.

We enrolled 15 nonsmoking adults to determine the feasibility of measuring personal exposure to ETS at work and to characterize workplace exposures of this small group of subjects. Indicators of exposure, measured during a workday, included questionnaires, personal samples for respirable particles (RSP) and nicotine, and urinary and salivary cotinine.

## Methods

Between October 1986 and May 1987, 15 nonsmoking volunteers (eight men, seven women), 18 years of age and

From the New Mexico Tumor Registry, Cancer Center (Coutas, Samet) and the Department of Environmental Science Engineering, Harvard School of Public Health (McCarthy, Spengler).

Address reprint requests to David B. Coutas, MD, Assistant Professor of Medicine, University of New Mexico Medical Center, New Mexico Tumor Registry, 900 Camino de Salud NE, Albuquerque, NM 87131. This paper, submitted to the Journal July 31, 1989, was revised and accepted for publication January 18, 1990.

© 1990 American Journal of Public Health 0090-0036/90\$1.50

older, were recruited from the Albuquerque, New Mexico area. We obtained exposure questionnaires, saliva, urine, and personal air particle samples during one workshift. The saliva and urine specimens were obtained before and after the workshift. Cotinine was quantitated by a double antibody radioimmunoassay, as described by Langone, *et al.*<sup>3</sup> Details of the assay in our laboratory have been reported previously.<sup>4</sup>

During the workshift, each subject wore a personal monitoring pump running at 1.7 l/min with a 10 mm nylon cyclone clipped to the shirt collar.<sup>5</sup> RSP samples were collected on 37 mm Fluropore filters (Millipore Corp). Nicotine was collected on a glass fiber backup filter treated with sodium bisulfate to minimize volatilization; after extraction from the filter, analysis for nicotine was done on a gas chromatograph with a flame ionization detector.<sup>6</sup> The recovery of nicotine by this procedure has been shown to be 98 percent efficient.

From the questionnaires, we derived measures of exposure including the total number of cigarette smokers and total number of hours exposed during the workshift. To describe the relationships among the measures of ETS exposure, Spearman correlations were calculated. Data analysis was performed with standard programs.<sup>7</sup>

## Results

Occupations of the subjects were diverse (Table 1); mean age was 44.8 years; average duration of the workshift and of the personal monitoring was 6.5 hours ( $SD = 2.0$ ).

Exposure to cigarette smokers at work was reported by 13 of the 15 participants. Of the 13 reporting exposure, two reported exposure to crowds of smokers during their workshift and the remaining 11 encountered a mean of 8.8 smokers ( $SD = 6.7$ ). The mean reported hours of exposure was 3.4 ( $SD = 2.1$ ).

Respirable particle and nicotine concentrations varied widely with the reported number of smokers and hours of exposure. The mean concentrations for RSP and nicotine were  $63.9 \mu\text{g}/\text{m}^3$  ( $SD = 41.5$ ) and  $20.4 \mu\text{g}/\text{m}^3$  ( $SD = 20.6$ ), respectively. Correlations between the atmospheric markers

2023380545

TABLE 1—Description of Participants in a Personal Monitoring Study of Exposure to Environmental Tobacco Smoke at Work, New Mexico, 1986–87

Occupation/Workplace	Workshift Duration/ Exposure Duration (hours)	RSP ( $\mu\text{g}/\text{m}^3$ )	Nicotine ( $\mu\text{g}/\text{m}^3$ )
<b>Males</b>			
Physician/Hospital	8.5	52.3	10.0
Social Worker/Office	8.0	44.0	2.5
Stock Broker/Office	8.2	69.4	7.2
Bus Boy/Restaurant	8.8	145.8	46.0
Maintenance Worker/Retail Store	8.9	85.2	6.9
Barber/Barber Shop	8.0	14.7	4.0
Barber/Barber Shop	8.4	145.8	13.7
Volunteer/Hospital	4.2	80.0	48.0
<b>Females</b>			
Interviewer/Public Transportation	3.2	4.0	0.0
Travel Agent/Office	2.6	85.7	50.0
Travel Agent/Office	8.4	62.1	46.7
Attorney/Office	8.0	63.3	5.9
Volunteer/Hospital	4.3	27.6	6.3
Volunteer/Hospital	4.3	25.2	4.7
Volunteer/Hospital	4.4	53.2	53.2

and the questionnaire measures of exposure to ETS were moderate (Table 2).

As was observed for the atmospheric markers, the post-workshift urinary and salivary cotinine levels varied widely with self-reported exposure. In comparison with pre-workshift levels, post-workshift levels were not consistently increased. The mean pre-workshift urinary and salivary cotinine concentrations were 31.8 ng/mg Cr ( $SD \pm 67.6$ ) and 2.9 ng/ml ( $SD \pm 5.0$ ), respectively. For the post-workshift levels, the corresponding values were 19.7 ng/mg Cr ( $SD \pm 43.2$ ) and 3.5 ng/ml ( $SD \pm 5.9$ ).

Spearman correlation coefficients were calculated to examine the relations among the questionnaire variables, the atmospheric markers, and urinary and salivary cotinine (Table 2). Moderate correlations were obtained for self-reports and cotinine levels, and nicotine levels and cotinine levels. However, RSP levels and cotinine concentrations were not correlated.

TABLE 2—Spearman Correlations between Various Measures of Environmental Tobacco Smoke at Work, New Mexico, 1986–87

Correlated Measures	N	r
<b>RSP (<math>\mu\text{g}/\text{m}^3</math>) with:</b>		
Nicotine	15	0.57*
Total number of smokers	15	0.44
Total hours of exposure	15	0.53*
Postshift urinary cotinine	14	0.08
Postshift salivary cotinine	11	-0.07
<b>Nicotine (<math>\mu\text{g}/\text{m}^3</math>) with:</b>		
Total number of smokers	15	0.62*
Total hours of exposure	15	0.54*
Postshift urinary cotinine	14	0.60*
Postshift salivary cotinine	11	0.46
<b>Postshift urinary cotinine (ng/mg Cr) with:</b>		
Total number of smokers	14	0.39
Total hours of exposure	14	0.57*
<b>Postshift salivary cotinine (ng/ml) with:</b>		
Total number of smokers	11	0.63*
Total hours of exposure	11	0.45

\* $p < 0.05$

## Discussion

The controversial effects of involuntary smoking in the workplace need further investigation. The conduct of such research would be facilitated by the development of unintrusive and accurate methods of exposure assessment. Alternative approaches include active and passive monitoring, biological markers, and questionnaires. We have shown that personal monitoring for tobacco smoke components can be accomplished in the workplace. However, many employers and employees would not participate in the study because of concern about the wearing of pumps.

Despite the small number of subjects studied in this investigation, objective evidence of exposure to ETS was obtained in various workplaces. The levels of RSP and nicotine were similar to those observed in other investigations.<sup>6,8–10</sup> However, few of these studies included information on the intensity and duration of exposure to ETS.<sup>10</sup>

We observed moderate positive correlations among the questionnaire measures of ETS exposure, the results of personal monitoring for RSP and nicotine, and measurements of urinary cotinine. Each of these types of measures provides a differing index of exposure to ETS.<sup>1</sup> The questionnaire measures that were used assess source strength, but concentrations of ETS are also influenced by room volume and ventilation. Nicotine is a specific marker of exposure to ETS, whereas RSP is nonspecific. Cotinine levels reflect nicotine exposure, but also are determined by timing of specimen collection<sup>10</sup> and uptake and metabolism. Thus, tight concordance among these broad indicators of exposure used in this study would not be anticipated.

Because of the differing characteristics of questionnaires, personal monitoring, and biological markers for assessing ETS exposure, no single method should be considered as optimal for studying the workplace. We recommend that assessment of ETS exposure in indoor environments should utilize multiple approaches to characterize short- and long-term exposures. In population studies, questionnaire measures of exposure offer the simplest approach with personal atmospheric markers and biologic markers providing methods for estimating the potential magnitude of misclassification of self-reported exposure.

## ACKNOWLEDGMENTS

The authors thank Dr. Helen Van Nunenakos for providing the reagents for the radioimmunoassay and Irene Walkiw for technical assistance in performing the assay.

Supported by a cooperative agreement, EPA CR811650, from the US Environmental Protection Agency. Dr. Coulas is recipient of an Edward Livingston Trudeau Scholar Award from the American Lung Association. Presented in part at Indoor Air Quality '89, sponsored by the American Society of Heating, Refrigerating and Air-Conditioning Engineers and the Society for Occupational and Environmental Health, April 1989.

## REFERENCES

1. US Department of Health and Human Services: *The Health Consequences of Involuntary Smoking: A Report of the Surgeon General*. (DHHS [CDC] pub. no. 87-4390). Rockville, MD: US Public Health Service, 1986.
2. National Research Council: *Environmental Tobacco Smoke: Measuring Exposures and Assessing Health Effects*. Washington, DC: National Academy Press, 1986.
3. Langone JJ, Gjika HB, Van Nunenakos H: Nicotine and its metabolites. Radioimmunoassays for nicotine and cotinine. *Biochemistry* 1973; 12:5025–5030.
4. Coulas DB, Howard CA, Peake GT, Skipper BJ, Samet JM: Salivary cotinine levels and involuntary tobacco smoke exposure in children and adults in New Mexico. *Am Rev Respir Dis* 1987; 136:305–309.

2023386546

2023380547

AU  
PUBLIC HEALTH BRIEFS

5. Turner WA, Spangler JD, Dockery DW, Colborn SD: Design and performance of a reliable monitoring system for respirable particulates. *J Air Pollut Control Assoc* 1979; 29:747-748.
6. Hammond SK, Lederman BP, Roche AC, Schatzkin N: Collection and analysis of nicotine as a marker for environmental tobacco smoke. *Atmospheric Environ* 1987; 21:457-462.
7. SAS Institute, Inc: *SAS User's Guide: Statistics*. Version 3 edition. Cary, NC: SAS Institute, Inc, 1985.
8. Spangler JD, Tosteson RD, Tosteson TD, Mape DT, Soczak ML: Personal exposure to respirable particulates and implications for air pollution epidemiology. *Environ Sci Technol* 1985; 19:700-707.
9. Mamatoku M, Uematsu S, Okuda T, Tomita H: Estimation of personal exposure to tobacco smoke with a newly developed nicotine personal monitor. *Environ Res* 1984; 35:219-227.
10. Marano ME, Boyd G, Byar D, Brown C, Callahan JF, Conde D, Cuttino JW, Granchi J, Haley NJ, Hammond K, Lewis J, Reeves W: Passive smoking on commercial airline flights. *JAMA* 1989; 261:867-872.

### EYE HOSPITAL EYE

SIR.—We read your Nov 5 editorial with special interest because we have recently experienced a large hospital outbreak of epidemic keratoconjunctivitis due to adenovirus type 8 which illustrates many of the points you emphasise. In one respect, however, our experience differed. Whereas you state that "the virus seldom spreads to family members" we found domestic transmission was frequent and serious. Of 161 virologically proven cases where an epidemiological history was available, 26 (16%) resulted from transmission within the family. This figure underestimates the true extent of domestic transmission since by no means all the secondary cases attended the eye hospital and so were not investigated virologically. Transmission to spouses, children, and grandchildren was recorded, and transmission was not restricted just to those relatives who helped to put drops into an infected person's eyes.

It is very important for every patient with possible adenovirus keratoconjunctivitis to be warned by the examining ophthalmologist, as early in the infection as possible, of the possibility of family spread. Patients should be told how the virus may spread from eye to eye in ocular secretions and advise on hygienic measures such as the use of separate towels and pillows slips, the use and careful disposal of paper tissues rather than handkerchiefs, and the need for thorough handwashing after the infected eye has been touched.

Whilst control of an epidemic depends, as you emphasised, on preventing the spread of virus within the eye hospital, effort should also be directed towards avoiding transmission at home. However, despite medical advice to an individual about hygienic measures, transmission may still occur within the family, because of the infectivity of the virus under conditions of close domestic contact.

North Manchester Regional Virus Laboratory,  
Bath Hall Hospital,  
Manchester M9 2AA  
Manchester Royal Eye Hospital

SHIRLEY RICHMOND  
CHRISTOPHER L. DODD

SIR.—Your editorial correctly indicates the dominant importance of hospitals and ambulance rooms in the spread of eye infections and the value of thorough washing with soap and water for preventing spread. Without decrying the value of hypochlorite and chloramine T, it is worth emphasising that the simple measure of thorough washing of hands and instruments with soap and running water can arrest hospital outbreaks.<sup>1</sup> Transmission within the home and in other non-hospital, non-industrial settings can also occur when hygiene is inadequate. Several examples of this were seen during epidemics of keratoconjunctivitis on Clydeside, with a secondary attack rate of 2% in family contacts surveyed during the 1971–72 outbreak.<sup>2</sup>

Department of Infectious Diseases,  
Communicable Diseases (Scotland) Unit,  
and Regional Virus Laboratory,  
Ruchill Hospital,  
Glasgow G20 9NB

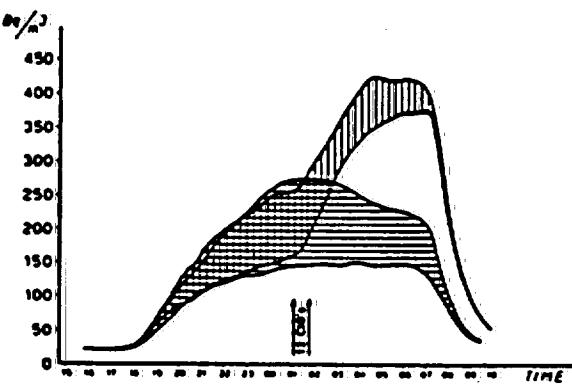
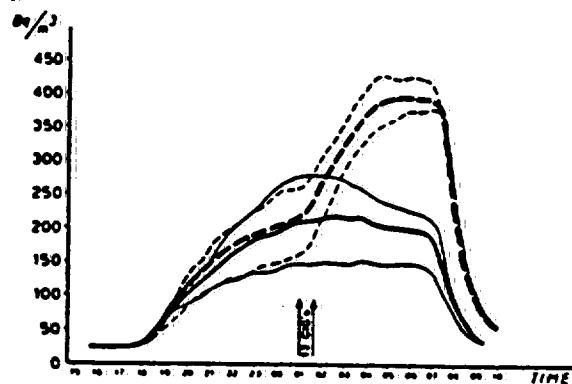
NORMAN R. GRIST  
DANIEL REID  
ELEANOR J. BELL

### ✓ PASSIVE SMOKING AND INDOOR RADON DAUGHTER CONCENTRATIONS

SIR.—Professor Correa and colleagues (Sept. 10, p. 595) have provided further evidence that passive smoking is a risk factor for lung cancer, but even more interesting was the association of lung cancer and maternal smoking only among smokers.

Observations about passive smoking should be considered against a background of naturally occurring radioactivity in dwellings—i.e., radon emanating into houses from the ground and from building materials.<sup>3,4</sup> The decay of radon results in  $\alpha$ -emitting radon

daughters, which attach to aerosol particles in indoor air,<sup>5</sup> such as cigarette smoke, and radon daughter exposure is a well-established risk factor for lung cancer among miners.<sup>6</sup> In support of this view, we wish to present some preliminary studies of indoor radon daughter concentrations as influenced by cigarette smoke. The accompanying figure shows the build-up of radon daughters in a room after the mechanical ventilation has been turned off and the additional concentrations obtained by burning cigarettes. Radon daughter concentrations seem to almost double. The instrument we used was a WLM-300 (EDA Instruments, Canada) and twelve cigarettes were passively burned in a room of 24 m<sup>3</sup>, with no-one present.



Mean and upper and lower curves for radon daughter concentrations in four experiments with (---) and three experiments without (—) cigarette smoke.

Mechanical ventilation was turned off at the beginning of experiment and started again at the end.

Stirring the air tends to cause the radon daughters to attach to walls and furniture and aerosols provide additional surfaces for attachment—i.e., there could be even greater differences in radon daughter concentrations in the absence and the presence of cigarette smoke under normal home conditions, when people move around causing some circulation of the air. Although we turned off the ventilation to increase the background of radon daughters and to facilitate the measurements, the concentrations found are by no means remarkable; the Swedish standard permits up to 400 Bq/m<sup>3</sup> EER (equilibrium equivalent radon), and thousands of homes have even higher concentrations.<sup>7</sup>

1. Wegman DH, Garner VE, Millett SJ. Epidemic keratoconjunctivitis. *Am J Public Health* 1970; 60: 1236-1237.

2. Read D, Bell RJ, Grist NR, Taylor JC, Ellis JR. Epidemic keratoconjunctivitis in the West of Scotland, 1967-72. *Trans R Soc Trop Med Hyg* 1974; 68: 157-61.

3. Cliff KD. Assessment of airborne radon daughter concentrations in dwellings in Great Britain. *Indust Biol Chem* 1978; 23: 709-711.

4. Strandén E, Norrby L, Uglem P. Alpha decay of radon in dwellings. *Health Phys* 1979; 36: 413-21.

5. Krueger J, Noshling JF. A comparison of the attachment of the decay products of radon-220 and radon-222 to mono-dispersed aerosols. *J. Aerosol Sci.* 1979; 10: 573-76.

6. Committee on the Biological Effects of Ionizing Radiations. The effects on populations of exposure to low levels of ionizing radiation (BEIR III). Washington, DC: National Academy of Sciences; 1980.

7. Åbergström G, Wilson C. Radon: geological aspects of an environmental problem. Uppsala Geological Survey of Sweden, 1982 report 10.

2023380548

The risk ratio for passive smoking can be surprisingly high (up to 2 or 3), as reported both by Cornes et al and others.<sup>1,2</sup> These risk ratios would be more consonant with those found for active smoking, particularly among women, if the active smoker is at greater risk also from his or her own passive smoke, again through the absorption of radioactivity on the smoke particles passively inhaled; also the relatively higher toxicity of the sidestream smoke might be important.<sup>1,3</sup> These and other aspects (e.g., the urban-rural difference in lung cancer risk from smoking) are more thoroughly discussed elsewhere<sup>2</sup> in the context of indoor radon daughter exposure.

Finally, in view of the long latency periods observed among mothers acquiring lung cancer from radon daughter exposure,<sup>1,4</sup> one might suggest that the children of smoking mothers obtain an early exposure to increased levels of radon daughters at home and that smoking later in life promotes the development of lung cancer.

Department of Occupational Medicine,  
University Hospital,  
S-61 01 Lund, Sweden

#### John Patterson RTTV Author

#### LUNG CANCER AND PASSIVE SMOKING

Sir,—I was surprised to read, in Professor Trichopoulos' and colleagues' letter (Sept 17, p 677), a German study of passive smoking and lung cancer described as having yielded "positive" results. The paper cited contains only relative conclusions based on poor data analysed by unacceptable methods.

I was also surprised that the findings from the Greek hospital study of passive smoking and lung cancer were almost identical to those reported two years ago,<sup>1</sup> despite a substantial increase in the numbers of cases and controls. In the 1981 report the relative risks of lung cancer for non-smoking women were 1, 1.6, 2.4, and 3.4 according to whether their husbands did not smoke, were ex-smokers, or were current smokers of 1–20 or 21 or more cigarettes a day; the updated relative risks are 1, 1.9, 2.4, and 3.4, respectively.

In the 1981 paper the relative risks agreed exactly with the appropriate cross-product ratios calculated from the numbers of cases and controls in the relevant category for husbands' smoking. In the latest results, despite the method being apparently identical, there is a clear disagreement between the relative risks provided by Trichopoulos et al and those I calculate (see table).

#### RELATIVE RISK OF LUNG CANCER ACCORDING TO SMOKING HABITS OF HUSBAND

Group			Cigarettes per day (former smokers)	
	Non-smokers	Ex-smokers	1–20	21+
RR (quoted)	1.0	1.9	2.4	3.4
RR (calculated)	1.0	1.9	1.9	2.5

Relative risk of risk of lung cancer among women whose husbands belong to a particular smoking category is the same among women whose husbands are non-smokers.

My calculations suggest that the latest data do not show as clear an association between a woman's lung cancer risk with her husband's smoking habits as the earlier data did. Indeed, relative risks calculated from the additional data are 1.2, 0.1.6, and 1.8 and do not show the dose-response relation seen earlier. This doubt, added

<sup>1</sup> Hartman T. Non-smoking wives of heavy smokers have a higher risk of lung cancer: a study from Japan. *Br Med J* 1981; 282: 619–621.

<sup>2</sup> Trichopoulos D, Kalathur A, Serrone L, MacMahon B. Lung cancer and passive smoking. *Am J Cancer* 1981; 147: 1–4.

<sup>3</sup> Supreme General. The health consequences of smoking. Cancer. Washington, DC: US Dept of Health, Education and Welfare, 1982; DHHS Pub 82-5077B.

<sup>4</sup> Scott SJ. Passive smoking and lung cancer. *Lancet* 1982; ii: 104–105.

<sup>5</sup> American Osteopathic Association. Lung cancer and smoking. *Am J Osteopathol Med* 1978; 81: 40–52.

<sup>6</sup> Kouch A, Bohn H, Schmid F. Passivrauchen ist Langzeitbedrohung für Kinder und Jugendliche. *Arbeitsmedizin* 1981; 10: 93–96.

<sup>7</sup> Trichopoulos D, Kalathur A, Serrone L, MacMahon B. Lung cancer and passive smoking. *Am J Cancer* 1981; 147: 1–4.

no doubt about the biological evidence and the use of cases and controls from different hospitals (limitations which Trichopoulos et al concede), prompts one to ask if the study really does add to the evidence implicating passive smoking as a factor in lung cancer.

Institute of Nutrition,  
University of Konstanz,  
D-7750 Konstanz 1, West Germany

Wolfgang Heller

#### POTASSIUM CHLORIDE SUPPLEMENTS

Sir,—As your Round the World correspondent predicted,<sup>1</sup> the US Food and Drug Administration advisory committee meeting of March 2 on the controversy of "slow-matrix versus microencapsulated potassium chloride preparations" proved inconclusive. A few points about this controversy are worth noting. The study by McKelahan et al,<sup>2</sup> showing a favourable result for "Micro-K" (A. H. Robins) in comparison with "Slow-K" (Giba-Geigy) was sponsored by Robins. The study by Patterson et al,<sup>3</sup> showing no difference between micro-K and slow-K, was sponsored by Giba-Geigy. Both studies have been confirmed by other studies sponsored by the respective company.

Giba-Geigy, while denying that slow-K is more ulcerogenic than micro-K, has bought from Alfred Benzon Ltd, Denmark, a licence for "Kalmorn," a microencapsulated (pellet) preparation of KCl similar (or identical) to micro-K. It seems remarkable that Giba-Geigy is planning to market this preparation when, according to Giba-Geigy's US subsidiary, "Slow-K has an established clinical record unparalleled by any other solid K supplement".

It seems that, privately, Giba-Geigy has concluded that Kalmorn is as good as micro-K, and that it is better than slow-K, but they would presumably consider it scientifically incorrect to conclude that micro-K is better than slow-K.

Finally I would emphasize, as your RTTV correspondent did, that doctors should "re-evaluate, as your RTTV correspondent did, that supplements and, if the indication is clear, prescribe it as a liquid". The findings of Patterson et al<sup>3</sup> clearly support this.

S. J. Patterson, Boston

Ole Hansson

Helsingborg, West Sweden

"This letter has been shown to Dr Burley, whose reply follows.—Ed. L."

Sir,—One of the main reasons why slow-release formulations of potassium were developed was the unacceptable liability of liquid potassium. Indeed, Patterson et al reported that KCP diet was poorly tolerated in their trial, giving rise to abdominal pain and diarrhoea in 9 of the 15 volunteers (60%). Dr Hansson omits to mention this. The issue is therefore whether the risk/benefit ratio of "Slow-K" is acceptable. There are eighteen years of clinical experience with slow-K in the UK, during which over 4.5 million patient-years of treatment has been prescribed; with "Micro-K" formulations there is almost no clinical experience. Less than 50 cases of significant alimentary side-effects have been reported with slow-K, and some of these were manifestly brought about by previous structures or oesophageal obstruction due to cardiac enlargement. It would be hard to point to a comparable safety record with any other widely used drug. The fact that a company may be investigating or pursuing alternatives is an indication of interest and involvement in the area, and should not be interpreted as a loss of confidence in an existing product.

Ciba-Geigy Pharmaceuticals,

Dennis Burley

<sup>1</sup> Adam P. Potassium supplements and upper gastrointestinal tract. *Lancet* 1981; i: 466.

<sup>2</sup> MacMahon MG, Alsalami K, Ryan J, Bunn A. Upper gastrointestinal and kidney side effects of potassium chloride supplements: a controlled clinical trial. *Lancet* 1981; ii: 1076–1079.

<sup>3</sup> Patterson DJ, Wrennen GS, Jeffries GM. Endotracheal intubation of adult and infant patients. *Am J Cancer* 1981; 147: 1–4.

2023380549

## INDOOR RADON DAUGHTER CONCENTRATIONS AND PASSIVE SMOKING

H. Bergman, C. Edling, and O. Axelson

Department of Occupational Medicine, University Hospital, Linköping, Sweden

(Received 26 February 1985; Accepted 4 November 1985)

Radon daughter exposure is a well-known cause of lung cancer in miners. Since radon daughters are known to attach to aerosol particles, it was also of interest to study to what extent radon daughters in indoor air might attach to cigarette smoke. Experiments were undertaken, which showed that moderate concentrations of radon daughters indoors could increase considerably and even more than double in the presence of cigarette smoke. The radon daughter levels obtained together with cigarette smoke may imply a risk of lung cancer for active and passive smokers.

### Introduction

Passive exposure to cigarette smoke has been associated with various nonmalignant diseases in humans and several studies also suggest passive smoking as a risk factor for lung cancer (Correa *et al.*, 1983; Hirayama, 1981; 1985; Trichopoulos *et al.*, 1981). Furthermore, in one of these studies, children of smoking mothers, i.e., passive smokers, who themselves take on smoking, were found to have an added risk of developing lung cancer later in life (Correa *et al.*, 1983).

Not only the toxic properties of various chemical components in the sidestream smoke are of interest, but also decay products of radon as present in indoor air, i.e. the radon daughters (radioactive isotopes of lead, bismuth and polonium). Radon emanates from soil, water and building materials and becomes dispersed in the air in the gaseous phase. One of the factors that influences indoor radon levels is plateout, the deposition of airborne radon daughters onto surfaces. This is affected by the ventilation rate, air filtration, deposition on walls and attachment to aerosol particles, cigarette smoke included (Bergman and Axelson, 1983; Jacobi, 1972; Jonassen and McLaughlin, 1982; Kruger and Nöthling, 1979; Porstendörfer *et al.*, 1978). Radon daughter exposure is a well-known risk factor for lung cancer among miners, both in the context of uranium mining and in other metal and iron ore mining (Axelson, 1982), and might therefore also

be suspected to impose a hazard to the general population through exposure in dwellings.

Radon daughters occur indoors in varying concentrations (Åkerblom and Wilson, 1982; Cliff, 1978; Strandén *et al.*, 1979). The levels can be quite high, sometimes even exceeding the standard permitted for exposure in mines. Indoor radon might escape from stony building materials, but particularly high indoor levels of radon and radon daughters usually depend on a leakage of radon from the ground into the houses, as revealed in a countrywide study in Sweden (Åkerblom and Wilson, 1982).

The aim of the present investigation was to further study the tendency of cigarette smoke to attract radon daughters, thereby increasing the airborne indoor radioactivity. Some quantitative aspects of the problem were studied also, i.e., the influence of the number of cigarettes smoked in various environments.

### Materials and Methods

The experiments on radon daughter concentrations and passive smoking were undertaken against a varying background of radon emanation, i.e., in three different localities: a room at the Department of Occupational Medicine, Linköping (lowest), a basement room in an apartment house and a room in a villa (highest).

The volumes of these rooms were about 30, 25 and 40 m<sup>3</sup>, respectively, and there were mechanical venti-

2023380350

lation systems automatically working in the daytime but not in the night hours in the first two localities but possible to regulate in the villa. Hence, the radon and radon daughter concentrations could periodically increase to a greater or lesser extent.

Between 2 and 12 cigarettes were passively burnt within an hour at a point in time when the radon daughters had reached a relatively stable concentration after the turn-off of the ventilation some hours earlier. The radon daughter concentrations (i.e., potential alpha energy concentration) were measured by a direct-reading instrument, WLM 300 (EDA Instruments, Canada), utilizing a filter and an alpha radiation detector. Readings of the radon daughter concentrations were obtained 10 times per hour throughout the time period under study. The results are given in  $\text{Bq}/\text{m}^3$  EER (equilibrium equivalent radon; ICRP, 1976), although simply referred to as  $\text{Bq}/\text{m}^3$ . This unit can be converted to working levels (WL) by dividing by 3700.

Stirring the air tends to cause the newly created, electrically charged radon daughters to attach more to walls and furniture, in a similar manner as they attach to aerosols. This phenomenon is referred to as the so-called wall effect. To account for this effect a small turbulence fan was used in some experiments to simulate the natural movements of the indoor air, needed because no persons were present in the rooms during the experiments. Each experiment was terminated with a turn-on of the ventilation.

## Results

The main results of the experiments are given in Figs. 1–4, where the build-up of radon daughters is shown in the absence and presence of cigarette smoke. One to five experiments were made under the same conditions, and the figures show the extreme curves

along with the averages in the absence and presence of cigarette smoke.

In the room with the lowest radon daughter concentrations, only a small absolute increase in radon daughters was obtained with the introduction of cigarette smoke (Fig. 1, the broken line), whereas the other experiments resulted in distinct increases in the radon daughter levels (Figs. 2 and 3). Only one experiment was done in the room of the villa but a quite clear increase in the concentration was obtained, i.e. from about  $700 \text{ Bq}/\text{m}^3$  to almost  $1500 \text{ Bq}/\text{m}^3$  with only four cigarettes (Fig. 4). Also in the room with the intermediate radon daughter level, about a doubling of the concentration took place after the burning of 12 cigarettes (Fig. 2). With the small turbulence fan present, the background radon daughter level became lower and the increase therefore relatively higher (Fig. 3). Furthermore, in one experiment (not shown) with the turbulence fan in operation, only 2 cigarettes in the room with the intermediate radon emanation raised the radon daughter level from a background of about  $100 \text{ Bq}/\text{m}^3$  up to about  $200 \text{ Bq}/\text{m}^3$ .

## Discussion

The conclusion from the results of these experiments seems to be that the indoor exposure to radon daughters may increase considerably in the presence of cigarette smoke, presumably because the newly formed, electrically charged daughters tend to attach to the smoke particles rather than to the walls, furniture, etc. The relative decrease in the concentrations, as obtainable with the turbulence fan and only in the absence of cigarette smoke, supports of this idea. The increase of radon daughters with increase of aerosol concentrations also are in accordance with other experiences (George *et al.*, 1983; Jacobi, 1972; Jonassen, 1984; Porstendorfer *et al.*, 1978).

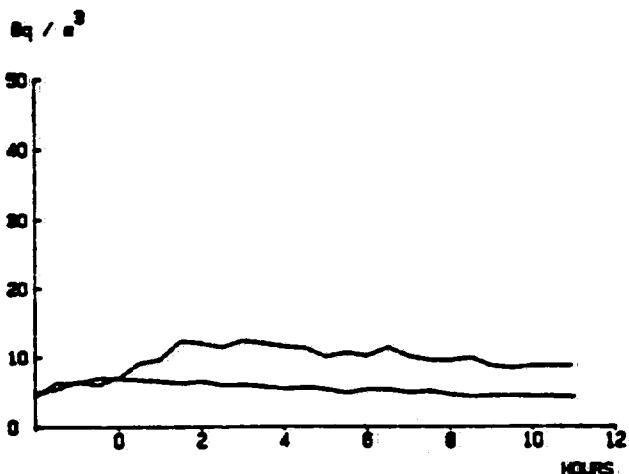


Fig. 1. Radon daughter concentrations in one experiment without (—) and three with (---) cigarette smoke. Twelve cigarettes burnt at 0. No fan in use.

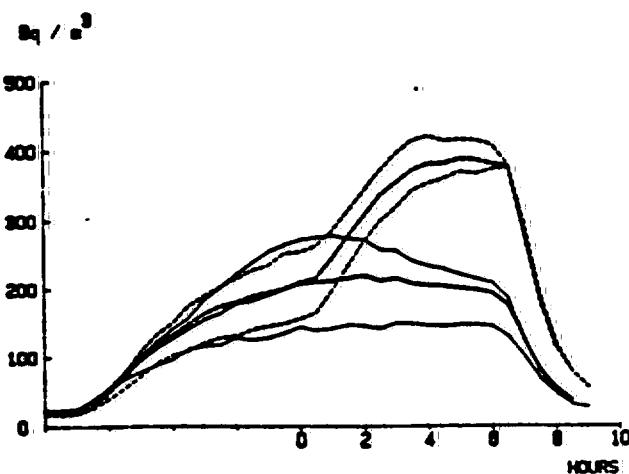


Fig. 2. Mean, upper and lower curves for radon daughter concentrations in four experiments without (—) and with (---) cigarette smoke. Twelve cigarettes burnt at 0. No fan in use.

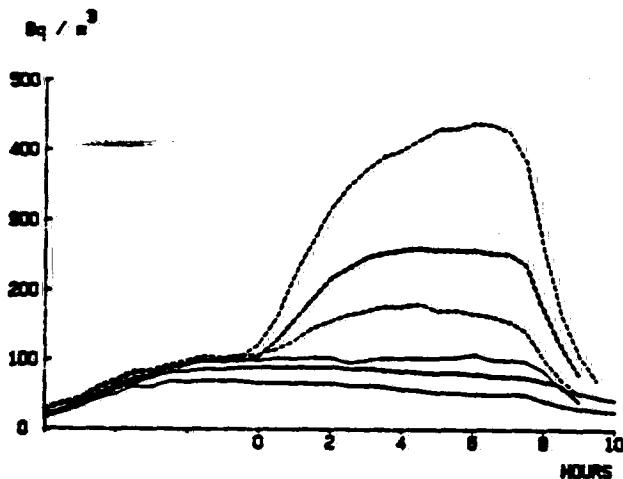


Fig. 3. Mean, upper and lower curves for radon daughter concentrations in five experiments with (—) and without (---) cigarette smoke. Twelve cigarettes burnt at 0. Fan in use.

However, cigarettes are also known to contain some amount of radioactivity, usually measured as polonium-210 (Radford and Hunt, 1964; Surgeon General, 1982). However, this radioactive content does not seem to play a significant role, as there was only a slight increase of the radon daughter levels in the presence of smoke but in the absence of any substantial radon emanation into the room.

The accuracy of our measurements might be questioned and unfortunately no particular comparison with other methods was possible. The results reported might therefore be valid only for those specific conditions under which they were obtained. However, our experience is that the results are reproducible, as indicated by the narrow range between the upper and lower curves in Fig. 2. Some uncertainty might exist with regard to the absolute concentration levels, but even so the readings obtained should be sufficiently correct in relative terms for the purpose of these experiments.

The biological importance of the relation of cigarette smoke and radon daughter exposure remains to be further evaluated, both with regard to the characterization of the airborne fraction of the daughter products and through epidemiologic studies. There are difficulties in assessing the relationship between exposure to radon and radon daughters, as measured by air sampling, and mathematical models (Stranden, 1979), as well as epidemiological approaches (Edling, 1983). On the basis of these experiments it can be concluded that the radiation risk resulting from the presence of radon in ambient air can be significantly increased as a result of presence of tobacco smoke.

## References

- Åkerblom, G. and Wilson, C. (1982) Radon: Geological aspects of an environmental problem. Report 30. Geological Survey of Sweden.  
 Axelson, O. (1982) Epidemiology of occupational cancer: Mining and

ore processing, in: Prevention of occupational cancer. Occupational Safety and Health Series No. 46, pp. 135-149. ILO, Geneva.

Bergman, H. and Axelson, O. (1983) Passive smoking and indoor radon daughter concentration, *Lancet* 8362, 1308-1309.

Cliff, K. D. (1978) Assessment of airborne radon daughter concentrations in dwellings in Great Britain, *Phys. Med. Biol.* 23, 696-711.

BEIR III (1980) Committee on the Biological Effects of Ionizing Radiations: The effects on populations of exposure to low levels of ionizing radiation. National Academy of Sciences, Washington, DC.

Correa, P., Pickle, L. W., Fontham, E. Lin, Y. and Haenszel, W. (1983) Passive smoking and lung cancer, *Lancet* 8362, 595-597.

Edling, C. (1983) Lung cancer and radon daughter exposure in mines and dwellings. Linköping University Medical Dissertations No. 157, Linköping, Sweden.

George, A. C., Knutson, E. O. and Tu, K. W. (1983) Radon daughter plateau—I. *Health Phys.* 45, 439-444.

Hirayama, T. (1981) Non-smoking wives of heavy smokers have a higher risk of lung cancer: A study from Japan, *Brit. Med. J.* 282, 183-185.

ICRP (1976) Radiation protection in uranium and other mines. ICRP publication No 24. Pergamon Press, Oxford.

Jacobi, W. (1972) Activity and potential energy of <sup>222</sup>radon and <sup>220</sup>radon-daughters in different air atmospheres, *Health Phys.* 22, 441-450.

Jonassen, N. (1984) Removal of radon daughters by filtration and electric fields, *Radiat. Prot. Dos.* 7, 407-411.

Jonassen, N. and McLaughlin, J. P. (1982) Air filtration and radon daughter levels, *Environ. Int.* 8, 71-75.

Kruger, J. and Nöthling, J. F. (1979) A comparison of then attachment of the decay products of radon-220 and radon-222 to monodispersed aerosols, *J. Aerosol Sci.* 10, 571-579.

Porstendorfer, J., Wicke, A. and Schraub, A. (1978) The influence of exhalation, ventilation and deposition processes upon the concentration of radon (<sup>222</sup>Rn), thoron (<sup>220</sup>Rn) and their decay products in room air, *Health Phys.* 34, 465-473.

Radford, E. P. and Hunt, V. R. (1964) Polonium 210: A volatile radioelement in cigarettes, *Science* 143, 247-249.

Stock, S. I. (1982) Passive smoking and lung cancer, *Lancet* 8361, 1014-1015.

Stranden, E. (1979) Population doses from naturally occurring radiation in Norway. SIS Report 1979:2. State Institute of Radiation Hygiene, Oslo, Norway.

Stranden, E., Bertheig, L. and Ugleverveit, F. (1979) A study on radon in dwellings, *Health Phys.* 36, 413-421.

Surgeon General (1982) The health consequences of smoking: Cancer. U.S. Department of Health, Education and Welfare, Washington, DC.

Trichopoulos, D., Kalandidi, A., Sparros, L. and MacMahon, B. (1981) Lung cancer and passive smoking, *Int. J. Cancer* 27, 1-4.

2023380352

## ENHANCEMENT OF EXPOSURE TO RADON PROGENY AS A CONSEQUENCE OF PASSIVE SMOKING

A. Alan Moghissi and Mary C. Seller  
Institute for Regulatory Science, Alexandria, VA 22307 USA

EI 87-402 (Received 5 November 1987; Accepted 1 May 1989)

Among indoor air pollutants, radon and tobacco smoke take dominant positions. Because radon decay products have a relatively short residence time in air, the extent of the equilibrium between radon and its daughter products is linearly proportional to the carcinogenic risk, at least at low exposure levels. The relevant factor is the equilibrium factor F. This paper discusses the enhancement of radon exposure as a result of the presence of particulate matter originating from tobacco smoke. The presence of tobacco smoke provides a mechanism for radon progeny to be attached to inhalable particles and to remain in indoor air for a prolonged time. The results of our study indicate a significant increase in F as a consequence of passive smoking. These modelling efforts are consistent with the experimental data reported previously.

### INTRODUCTION

The potential risks associated with radon exposure and its progeny have been known for several decades. Much of the current knowledge has been gained from the study of uranium miners. There have been numerous reviews of uranium miners who worked in the Erz Mountains and in Joachimsthal (both in Europe), in the Colorado Plateau in the U.S., in Canada, and in several uranium and other mines. The risk estimates for radon derived from these data require an extrapolation from high-exposure levels, common in mines during the period of study, to environmental levels. The problem is aggravated by insufficient knowledge of radon levels in the mines, the level of smoking among the miners, the exposure to other carcinogenic substances, including carcinogens and promoters in

the mine, and other activities of the miners. In spite of this, the results of the risk estimates, as performed by various investigative groups, are remarkably similar. The International Council on Radiological Protection (ICRP) has provided risk factors for radon exposure based on the models developed by Jacobi (ICRP 1987). These estimates deviate somewhat from those provided by the National Council on Radiation Protection and Measurements (NCRP 1985) which relied on the model developed by N. Harley. The National Academy of Sciences provided risk estimates in its BEIR III report (NRC 1980) and again later in its BEIR IV report (NRC 1988). The uncertainties of risks of exposure to radon can be assessed by comparing the risk estimates provided by these organizations. For example, the BEIR IV estimate is

lower by a factor of 2.1 than the BEIR III, and higher by a factor of 2.7 than the NCRP estimates. Despite the available data, none of these groups has attempted to provide a statistical uncertainty associated with the risk estimate.

In the past it was assumed that natural radioactivity was inevitable and thus acceptable (FRC 1960). Although radon measurements in indoor air were known for some time (Haque et al. 1965), the high radon concentrations measured in homes built over uranium mill tailings (Culot and Schiager 1973) drew attention to the potential risks associated with radiation exposure emitted from naturally occurring radionuclides. Despite the comparatively large radiation exposure from natural sources, many industrialized nations insisted upon substantial expenditures to reduce radiation exposure originating from industrial activities while failing to recognize the necessity to control the total radiation exposure. Regulatory agencies in the U.S. have recognized only recently the significance of radon as an important source of population exposure despite the fact that high levels of exposure were predicted (Moghissi and Carter 1974; Moghissi and Carter 1976).

The importance of tobacco smoke as a causative agent for the significant increase in the lung cancer rate of the population has been recognized for some time (Terry 1964). The number of studies dealing with tobacco smoke and its impact on human health is large and includes numerous critical reviews. Since the impact of smoking on human health is not the subject of this paper, it will not be discussed any further. The significance of passive smoking as a potential carcinogenic risk has been only recently recognized. The National Academy of Sciences (NRC 1986) evaluated the significance of environmental tobacco smoke (ETS) and provided evidence that ETS poses an adverse human health risk. Repace and Lowrey (1985) evaluated the epidemiological information on ETS and concluded that ETS caused a major portion of the lung cancer in the nonsmoking population. These authors also provided measured data on the concentration of particulate matter in several commercial establishments (Repace and Lowrey 1980).

This paper attempts to demonstrate that ETS enhances radon exposure. This enhancement is demonstrated to result in an enhancement of the risk of radon exposure.

#### RADON DOSIMETRY

There have been numerous studies dealing with radon dosimetry. These are included in ICRP (1987),

NRC (1988), NCRP (1985), and numerous other reports. The choice of the dosimetric model has no significant impact on the outcome of the results of this study. One possible area of concern is the impact of the unattached fraction of radon progeny. Certain models, notably the ICRP (1987), assign a higher risk to the unattached fraction of air as compared to that attached to comparatively larger particles.

It is well known that the radiation dose delivered to the lung is caused by the deposition of radon progeny, notably  $^{210}\text{Po}$ ,  $^{214}\text{Pb}$ ,  $^{214}\text{Bi}$ , and  $^{214}\text{Po}$ . The traditional method of evaluating radon exposure is based on the working level, WL, defined as 100 pCi Rn/L of air in secular equilibrium with its progeny. The SI equivalent for WL is J/m<sup>3</sup> and 1 WLM corresponds to 0.0035 Jh/m<sup>3</sup>. Because radon is seldom in complete equilibrium with its progeny, the relevant equilibrium factor is F, defined as the ratio of Rn with its daughter products:

Although it has been known that F depends strongly upon the presence of particulate matter in air, it has been mostly assumed that the concentration of particles is usually constant in indoor air and these F-values of 0.5 have been assumed conservatively (Haque et al. 1965). However, the recent acceptance of indoor air as a major source of exposure to air pollutants has directed attention to the enhancement of the impact of one pollutant as a consequence of the presence of others.

#### VARIATION OF F WITHIN INDOOR AIR

The literature is voluminous on the relationship between the presence of particulate matter and the F-value. Swedjemark (1983) developed appropriate equations based on theoretical studies conducted by Wicke (1979) and Porstendoerfer and Mercer (1978). George et al. (1983), Jonassen and McLaughlin (1982), and Porstendoerfer et al. (1978) published results of their studies on the various parameters impacting the F-factor. Many of these studies are based on the initial model developed by Jacobi (1972).

The conceptual design of all of these studies is based on the following principles. Radon is released from the soil or building materials at a constant rate and radon daughters are produced in accordance with the radioactive decay. Because these decay products are charged and are monoatomic, they are rapidly deposited on available surfaces. Accordingly, the deposition rate is proportional to the surfaces available in indoor air. The exchange of indoor air with fresh air removes the radon and particulate matter, and the removal rate is proportional to the air exchange rate. Instead of relating the deposition of radon daughters

2023380354

to the available surfaces, the number of particles per mL of air may be used as a substitute for the surface. Given the large uncertainties associated with modelling and the experimental set-up, this approach seems to be appropriate.

#### STUDY APPROACH

The objective of our study is to evaluate the potential enhancement of the F-value as a consequence of ETS presence in indoor air. Our study is based on Swedjemark's equations (1983). We chose the extremes of the air exchange rate (0.2 to 2) and the particulate matter to demonstrate the impact of ETS. We assumed particles associated with tobacco smoke do not have unique properties that impact the deposition of radon progeny. Similarly, we have not chosen a typical value for the concentration of tobacco smoke because such a concentration depends upon a variety of parameters which are site-specific.

#### RESULTS AND DISCUSSION

Fig. 1 contains the results of the modelling efforts. The F-values are computed for air exchange rates

ranging from 0.2 to 2 per hour and appropriate concentrations of particulate matter.

For low air exchange rates, the presence of particles significantly increases the F-value. As the air exchange increases, the presence of particles has less of an effect on the F-value. It can be seen that the F-value is significantly enhanced by the presence of particles originating from tobacco smoke. The results of our study are in agreement with the findings of Bergman et al. (1986), who experimentally demonstrated a five-fold increase in the F-value as a result of the presence of tobacco smoke.

One area of concern is the unattached fraction of Rn and the impact of particles as well as air exchange on the possible removal of the unattached fraction of Rn progeny. Because the attachment rate of the unattached fraction is significantly higher than other parameters, it is unlikely that the results of this study will be appreciably impacted by the consideration of unattached fractions of Rn.

#### CONCLUSIONS

It appears that ETS presents a greater risk than anticipated. In addition to its own risk, it may also

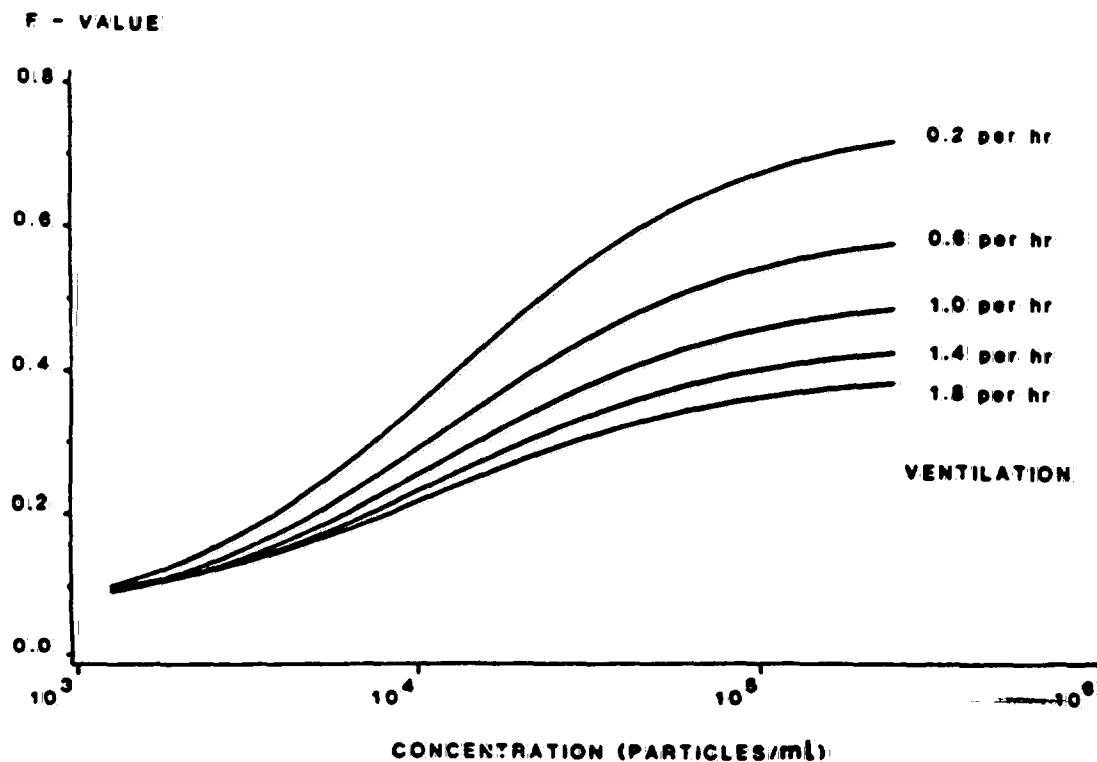


Fig. 1. F-values as a function of concentration and ventilation.

20233860555

be a significant enhancer of radon risk. On one hand, a clear distinction must be made between synergistic effects of smoking and radiation and, on the other hand, enhancement of radiation exposure. The synergistic effects of (active) smoking and radon exposure relate to cellular effects of combined exposure to carcinogens present in tobacco smoke and radiation exposure originating from radon progeny. In contrast, the radon exposure enhancement relates to the increased exposure to radon progeny as a consequence of passive smoking. In this latter case, tobacco smoke is a vehicle for the transport of radon daughters to the human lung. Repace and Lowrey (1980) have measured concentrations of tobacco tar ranging from 0.1 to 1 mg/m<sup>3</sup> in indoor air. Assuming a specific gravity of 0.8 and a median diameter of 0.5 μm, one can readily calculate that a concentration of 10<sup>6</sup> particles/mL is not too extreme. Therefore, an enhancement factor of 5 is not unreasonable. The "smoke-filled room" is not a healthy place and may pose a significant radiation risk.

**Acknowledgment**— This research was supported by a grant of the U.S. Department of Energy to the Institute for Regulatory Sciences. The views expressed in this paper are those of the authors and do not necessarily represent the views of the U.S. Department of Energy.

## REFERENCES

- Bergman, H.; Edling, C.; Asplund, O. Indoor radon daughter concentrations and passive smoking. *Environ. Int.* 12:17-19; 1986.]
- Cubot, M.V.J.; Schiager, K.J. Radon progeny control in buildings. Fort Collins, CO: Colorado State University; 1979
- FRC (Federal Radiation Council). Background material for the development of radiation protection standards. FRC Report No. 1. Washington, DC: Federal Radiation Council; 1960.
- George, A.C.; Knutson, E.O.; Tu, K.W. Radon daughter plateau. *J. Health Phys.* 45:439-444; 1965.
- Haque, A.K.; Collinson, A.J.L.; Blyth Brooks, C.O.S. Radon concentration in different environments and the factors influencing it. *Phys. Mod. Biol.* 10:505; 1965.
- ICRP (International Commission on Radiological Protection). Lung cancer risk from indoor exposures to radon daughters. New York: Pergamon Press; 1987.
- Jacobi, W. Activity and potential energy of radon-222 and radon-220 daughters in different air atmospheres. *Health Phys.* 22:441-450; 1972.
- Jonasson, N.; McLaughlin, J.P. Air filtration and radon daughter levels. *Environ. Int.* 8:71-75; 1982.
- Moghissi, A.A.; Carter, M.W. Consideration of background in the development of environmental standards. Proceedings of the Eighth Midyear Topical Symposium of Health Physics Society Conference 741018, Knoxville, TN; 1974; p. 335-360.
- Moghissi, A.A.; Carter, M.W. Natural and manmade radiation: is there a distinction? Proceedings of the Tenth Midyear Topical Symposium of Health Physics Society, Rensselaer Polytechnic Institute, Troy, NY; 1976; p. 334-339.
- NCRP (National Council on Radiation Protection). Evaluation of occupational and environmental exposure to radon daughters in the United States. Bethesda, MD: National Council on Radiation Protection and Measurements; 1985.
- NRC (National Research Council). The effects on population of exposure to low levels of ionizing radiation. Washington, DC: National Academy Press; 1980.
- NRC (National Research Council). Environmental tobacco smoke - measuring exposures and assessing health effects. Washington, DC: National Academy Press; 1986.
- NRC (National Research Council). Health risks of radon and other internally deposited alpha-emitters. BEIR IV. Washington, DC: National Academy Press; 1988.
- Porstendorfer, J.; Wicke, A.; Schraub, A. The influence of exhalation, ventilation and deposition processes upon the concentration of radon, thoron and their decay products in room air. *Health Phys.* 34:465-473; 1978.
- Porstendorfer, J.; Mercer, T.T. Influence of nuclei concentration and humidity upon attachment rate of atoms in the atmosphere. *Atmos. Environ.* 12:2223-2228; 1978.
- Repace, J.L.; Lowrey, A.H. Indoor air pollution, tobacco smoke and public health. *Science* 208:464-472; 1980.
- Repace, J.L.; Lowrey, A.H. A quantitative estimate of nonsmokers' lung cancer risk from passive smoking. *Environ. Int.* 11:3-22; 1985.
- Swedjemark, G.A. The equilibrium factor F. *Health Phys.* 45:453-462; 1983.
- Terry, L.K. Smoking and health. Washington, D.C.: U.S. Department of Health, Education and Welfare; 1964.
- Wicke, A. Studies on the natural radioactivity in air in living areas: Basic studies for the estimation of radiation exposure from radon and thoron decay products. University of Giessen, Federal Republic of Germany; 1979. Dissertation.

2023380556

## CORRESPONDENCE

### Indoor Radon and Lung Cancer in China

We read with great interest the recent paper by Biot et al. (1). Their study was an elegant technical application of a case-control design to an important question of the relationship between residential radon exposure and lung cancer. The case-control design is certainly appropriate for investigating this relationship. However, there are several important problems that would preclude the detection of radon-induced lung cancer in this population.

First, the choice of a population with "exceptionally high rates of lung cancer," but without exceptionally high radon levels (mean, 2.3 pCi/L; 20% of levels exceed 4 pCi/L), is curious. Clearly, one would not expect many of the excess lung cancers in this population to be attributed to radon. In fact, one would anticipate, *a priori*, that the effect due to radon would most likely be obscured.

The high levels of other indoor air pollutants, particularly benzo[a]pyrene at levels 70 times higher than in the U.S., would provide a biologically plausible alternative hypothesis for the excess malignancies. As the authors state, "confounding by other risk factors may have hindered detection of increased risks among women exposed to high radon levels." Certainly, the excess malignancies due to such risk factors would dilute the ability to detect radon-induced malignancies, unless there was a significant positive interactive factor.

Second, the consistently high, indoor dust levels will have significantly reduced the exposure dose of the respiratory epithelium to alpha radiation. The unattached fraction of radon daughters will decrease in this setting, lowering the dose of radiation to the respiratory tract for any given exposure level measured by alpha-track. The number of radon-induced lung cancers for a given ambient exposure level could be significantly reduced by this

effect. The authors state that they attempted to adjust for this effect by comparing lung cancer rates at high and low indoor air pollution levels. However, uncertainties relating to this adjustment render it meaningless because (a) even the lower air pollution categories were dusty by western standards, (b) as dust levels increase, there is an increase in exposure to other carcinogens, which would be expected to cause an increase in non-radon-induced lung cancers, tending to offset any decrease in radon-induced lung cancers associated with increasing dust levels, and (c) there is inadequate information about dust particle size distribution to predict the relative dosimetric effect of the authors' categories of dust exposure.

A third problem, common to all current epidemiologic investigations of residential radon, is the uncertain relationship between currently measured levels and lifetime exposure doses. This uncertainty, even when using year-long alpha-track measurements, creates a bias to underestimate the effect of radon due to potential misclassification of exposure.

It is interesting, despite all of these problems (low exposure levels, a further dosimetric reduction of exposure due to dust, exposure to other lung carcinogens, and uncertainty in estimating historic exposure), that a slight upward trend for small cell carcinoma of the lung was nonetheless linked with increasing dose. As the authors point out, it is this histologic form of lung cancer that has been disproportionately increased in other radon studies (2-4). The importance of this finding must be adequately stressed.

Although the authors recognize some of the noted limitations of this study, additional caution must be applied. It would be inappropriate to suggest, on the basis of this study, that indoor-radon lung cancer risk has been overestimated. Further, readers must be advised that this study provides no justification for raising the remedial action level of 4 pCi/L suggested by the Environmental Protection Agency. The press is closely following and reporting on radon investigations and cautions to avoid overinterpretation of the data are incumbent upon scientists in the field!

MARK J. UPFAL\*  
Occupational Medicine  
Wayne State University  
School of Medicine  
Detroit, Mich  
G. ALEX JOHNSON  
American Lung Association of  
Southeast Michigan  
Southfield, Mich  
A. P. JACOBSON  
Radon Resource and Training Center  
PEGGY A. BRADY  
University of Michigan  
School of Public Health  
Ann Arbor, Mich  
JOHN A. CAMPBELL  
Wayne State University  
School of Medicine  
Detroit, Mich

### References

- (1) BIOT WJ, XU ZY, BOICE JD JR, ET AL: Indoor radon and lung cancer in China. *J Natl Cancer Inst* 82:1025-1030, 1990
- (2) SAMET JM: Radon and lung cancer. *J Natl Cancer Inst* 81:745-757, 1989
- (3) DAMBER L, LARSSON LG: Combined effects of mining and smoking in the causation of lung carcinoma. A case-control study in northern Sweden. *Acta Radiol* 21:305-313, 1982
- (4) GOTTLIEB LS, HUSEN LA: Lung cancer among Navajo uranium miners. *Chest* 81:449-452, 1982

### Response

The correspondence by Upfal et al. raises the question of whether our case-control study (1) could evaluate, with any precision, the risks of lung cancer associated with indoor radon in Shenyang, China. Particular concern is expressed that the dustiness of homes in Shenyang may have influenced radiation dose to lung tissue. Although this issue could not be directly evaluated, it seems likely there was substantial variation in tissue exposure among the women studied because the differences in home air radon levels were so great. The median radon levels were 7.6 times greater in the high versus low exposure groups. Further, even dustier condi-

\*Correspondence to: Mark J. Upfal, M.D., MPH, Division of Occupational and Environmental Medicine, UHC-41, Wayne State University School of Medicine, 4201 St. Antoine, Detroit, MI 48201.

2023380557

archives of  
**Environmental Health**

An International Journal

March/April 1988

Volume 43 Number 2

First International Symposium  
on Environmental Epidemiology

June 3-5, 1987

**EPIDEMIOLOGY IN  
ENVIRONMENTAL HEALTH**

Sponsored by

the Center for Environmental Epidemiology, University of Pittsburgh  
and the United States Environmental Protection Agency

Graduate School of Public Health, University of Pittsburgh  
130 DeSoto Street, Pittsburgh, Pennsylvania 15261

6000 Albemarle Street, N.W.  
Washington, D.C. 20016  
Return Postage Guaranteed

ISSN 0890-432X

2023380558

## Contamination of Individuals by Radon Daughters: A Preliminary Study

JAMES H. STEBBINGS, Sc.D.  
JAMES J. DIGNAM, B.A.  
Environmental Health Section  
Biological, Environmental, and Medical Research Division  
Argonne National Laboratory  
Argonne, Illinois

**ABSTRACT.** Body radon daughter contamination reflects relative individual respiratory exposures to radon daughters; counts can be related both to household radon levels and to lung cancer risk factors such as sex and tobacco smoking. Radon daughters were counted by gamma spectroscopy from 180 adult residents of eastern Pennsylvania. A seven-position, 35-min scan was conducted in a mobile body counter, generally during afternoon or evening hours. Track-etch detectors for household radon were distributed, and were recovered from 80% of the subjects. Over 75% of the population had environmentally enhanced radon daughter contamination. House radon levels were strongly related, as anticipated, to radon daughter contamination in the 112 subjects for whom both sets of measurements were available ( $p < .001$ ); basement measurements were as strongly related to personal contamination as were living area measurements; bedroom measurements were slightly more strongly correlated. Both sex ( $p < .02$ ) and cigarette smoking ( $p < .01$ ) significantly modified the relationships, after nonlinear adjustment for travel times. Using a logarithmic model, a given house living-area radon level was associated in females with body contamination by radon daughters 2-3 times that in males. Nonsmokers had 2-4 times higher levels of contamination than smokers. Results are for the total of internal and external contamination, these being highly correlated in preliminary experiments. Time usage and activity patterns of the subjects are believed to be important in explaining these findings, and may become important variables in radon risk assessment.

ACTIVE TOBACCO SMOKING causes the majority of cases of lung cancer in industrialized societies; passive tobacco smoking and radon daughters are believed to be the next most important environmental risk factors for lung cancer. Several case-control studies of lung cancer in relation to passive smoking and radon are now underway.

Concurrently, extensive public health programs and private commercial activities directed toward the measurement of radon in homes, with an aim toward re-

mediation, have arisen in response to the recognition that large regions of the United States average higher radon levels than previously recognized.<sup>1-3</sup> There are areas, such as the Reading Prong of Pennsylvania, which are extreme.

Neither in the case of passive smoking<sup>4</sup> nor in the case of radon is the evidence firm regarding the real importance of the problem in the general population; significant epidemiological studies of radon daughters are all of miners exposed occupationally.<sup>5</sup> Further, estima-

tion of the numbers of lung cancer cases to be expected, given published dose-response relationships and reported radon levels in homes; ignores complications resulting from population heterogeneity in age, sex, smoking habits, residential patterns, and personal time-usages. The importance of these factors is currently uncertain, although attention has been paid to interactions of tobacco smoking and radon. In addition to the possibility of biological synergism between radon daughters and tobacco smoke,<sup>5</sup> there is believed to be a positive dose interaction between the two,<sup>6,7</sup> and population smoking habits are critical in the application of radon risk assessment models.<sup>8</sup> The positive dose interaction may translate into a doubling of radon daughters in the house atmosphere given a certain radon level, if tobacco is being smoked.<sup>9</sup> This neglects questions of particle size distributions and of the unattached fraction.

In this paper we present an adventitious study that carries empirical measurement of radon daughter exposures of persons in households closer to the desired endpoint, bronchial epithelial dose, than do measurements of radon in home atmospheres. Specifically, we relate the combined internal and external contamination of individuals by radon daughters at a point in time to long-term average radon levels in the home and to the personal characteristics of active smoking status and sex. Although we would have preferred to measure internal contamination levels only, we assume that total contamination directly reflects radon daughter levels in the breathing zone of individuals. Repeated point estimates of radon daughter contamination would also have been preferable to the single-point estimates available to us.

We are not proposing our methodology as a generally useful approach in studies of the health effects of radon. We claim only that some empirical studies relating house measurements of radon to exposures of individuals in real populations, accounting for as many variables as possible which may mediate bronchial dose given house radon levels, are desirable. The existence of models, however much confidence we have in them, cannot be an argument against the collection and analysis of empirical data in the absence of the latter. Only a plenitude of empirical data is an argument against collecting further empirical data.

## Methods

These data are from a study of radium contamination in an occupational cohort employed in the dial painting industry in the post-World War II era. No detectable occupational burdens were found. Radon is a decay product of radium, and the 1.76 MeV gamma ray from the decay of the radon daughter, <sup>210</sup>Bi is used to measure radium in the body.

**Population.** The U.S. Radium Company moved its radium dial painting operation to a community in northeastern Pennsylvania in the late 1940s. Radium was used for two decades. An employee roster was acquired, and some subjects with potentially high exposure to radium were counted at Argonne National Lab-

oratory. In late 1983 and early 1984 a field effort was made to count remaining subjects. Subjects living within a 60-mile radius of the community, not previously measured at Argonne, and for whom a radiation exposure record from prior to 1969 was available (some additional records were later located), were invited to our mobile body counter for measurement. Approximately 65% of the invited subjects were measured.

Of 232 individuals measured, 52 have been excluded from these analyses: 11 were controls or family members, 1 was incompletely measured, 17 were measured after the heating season (after 1 April 1984), and 23 still worked in buildings with radium/radon contamination. After exclusions, there were 180 subjects for analysis: 4 were co-residents of homes with other subjects.

A questionnaire was administered regarding activities of the day, time since leaving home, current smoking habits, and occupation. Height and weight were measured (with shoes and jackets off). Nearly all measurements were performed during afternoon or evening hours.

**House radon measurements.** Alpha-track detectors manufactured and read by the Terradex Company were distributed to subjects when they visited the mobile body counter. A questionnaire concerning house characteristics was administered during the count, and subjects were instructed on placement of detectors (away from floor and outside walls, away from direct drafts). Generally, three detectors were distributed: one each for the basement, the main bedroom, and for the room on the first floor where most family activity occurred. Actual room locations were coded, but have been analyzed as basement, first floor, and main bedroom measurements. Track-etch detectors were placed for an average of 10 months (range: 1.9 to 15.1 mo; only two measurements were for less than 6 mo). Due to supply difficulties, detectors were actually sent with only 155 of the 180 subjects reported on here. Of these 155 subjects, 124 returned the detectors. For these 124, 112 results from main floors were available.

In this paper we related body counts to main floor living area measures because most body counts were in afternoon and evening hours. Conclusions based on basement or bedroom measurements do not differ significantly.

**Body counting.** Subjects lay on their backs in a lead-shielded bed movable under a fixed 8"-diameter NaI crystal approximately 39 cm above the dorsal (back) surface of the subjects. Five-minute counts were made at mid-body (50%), and at 35%, 20%, 5%, 65%, 80%, and 95% of body height, in that sequence (mid-body to feet to mid-body to head). Late in the study period the sequence was modified to begin at position 65%, repeating that position in the original sequence. Forty-two subjects were measured in this way; statistical analyses indicate that this change did not bias results presented here. As time was required to adjust the bed and reinitialize the instruments, approximately 49 min, or 7 min per position, were required to complete the procedure. The 1.76 MeV gamma ray from the decay of <sup>210</sup>Bi in and on the body was counted to measure radium. Background measurements, without a subject in

the bed, were taken at least twice a day; the background for an individual was computed as the mean of the preceding and following background counts. Details of procedures for radium measurement at Argonne have been published.<sup>10</sup>

The index of body contamination by radon daughters analyzed for these preliminary analyses was the sum of the counts at each of the seven positions minus background, yielding a total counts per minute (CPM). Principal components analyses (unpublished) suggested that approximately equal weightings of the seven positions is not inappropriate. A mean internal radium calibration factor for the seven position scan is  $0.87 \pm 0.11$  nCi  $^{214}\text{Bi}$  per CPM above background.

Two potential confounders were time since leaving home, which relates to decay of radon daughters and radon excretion, and body size, which relates to counting geometry. Decay of unsupported radon daughters in the thorax and of daughters supported by radon in the body, after leaving a house with high radon levels, was described by Rundo et al.<sup>11</sup> The content of  $^{214}\text{Bi}$  (RaC) in a mixture consisting initially of equal activities of RaA, RaB, and RaC is reduced to half in about 1 hr, and is considerably longer than the longest single half-life—26.8 min—for RaB. The biological excretion of radon is described by a multicompartment exponential model.

We experimented with adjustment of observed counts to time zero by backwards extrapolation, utilizing either the decay curve of RaB or an empirical decay curve estimated from higher burden subjects with double measurements 30 min apart at position 3 (65% of height). Such adjustments do not qualitatively change the results presented below, but considerable variance is introduced into the data. Many measurements at longer travel times are near zero; back extrapolation magnifies random error.

The approach chosen was to introduce travel time ( $T$ ) and its square as independent variables into the prediction equation for CPM. This approach yields similar results in all models, but it has theoretical appeal only when the logarithmic transformation of CPM is used as the model

$$\ln \text{CPM} = b_0 + b_1 T + b_2 T^2 \dots$$

in antilog form is:

$$\text{CPM} = e^{b_0} \times e^{b_1 T} \times e^{b_2 T^2} \dots$$

where  $b_1$  and/or  $b_2$  may be negative.

Median travel times to initiation of the body count were 37 min; the 25th percentile was 24 min, the 75th, 76 min. As 7 min was the average time required for counting one position plus adjusting the bed position and reinitializing the instruments, we have added 21 min to the start to yield an approximate mid-time of 58 min from leaving home. Results presented below are adjusted to  $T = 1$  hr to avoid extrapolating beyond the observed data. In this time range, similar results are obtained from adjustments using empirical decays and methods that use time as an independent variable.

Body thickness modifies counting efficiency. Utilizing a formula for effective body thickness

$$d' (\text{cm}) = 22.5 \times (W/H)^{0.8}$$

(weight [W] in kg, height [H] in cm) derived from studies in an arc position,<sup>10</sup> little effective variation in body thickness was found. Mean  $d'$  was 15.2 cm; the 25% and 75% quantiles, 14.0 and 16.2; the 5% and 95% quantiles, 12.6 and 17.4. The range of mean  $d'$  among smoking categories was 14.9 to 15.4; between the sexes it was 15.1 to 15.5. In these analyses weight was used to correct for body size.

**Statistical methods.** Data were analyzed using SAS<sup>12</sup> multivariate procedures. The general model was

$$\begin{aligned} \text{CPM} = & b_0 + b_1 T + b_2 T^2 + b_3 Rn + b_4 Rn \\ & \times \text{sex} + b_5 Rn \times \text{smoking} + \dots \end{aligned}$$

where CPM is the sum of counts per minute for body positions 1-7,  $T$  is time from leaving home to mid-count,  $Rn$  is the first floor household radon level over the 6 to 12 mo immediately following, and sex and smoking are dummy variables taking the value 0 or 1 and entered only as interaction terms with radon. Sex and smoking terms are neither meaningful nor statistically significant in themselves. Additional variables investigated include passive smoking, height, weight, weight to height ratio, and measurement sequence. In nearly all instances regressions were run utilizing untransformed counts (which include some negative values due to background subtraction), the logarithmic transformation of counts (ln CPM if CPM > 1; 0 otherwise); and square root transformations of the absolute value of the counts, with the sign replaced. For the logarithmic equation house radon levels were defined as ln ( $Rn + 1$ ), but terms in  $T$  were not transformed. Qualitatively, all models yielded similar results, as do models with 1-3 outliers as defined by Cook's D<sup>13</sup> deleted.

## Results

The highly skewed frequency distributions of radon levels in homes of study subjects are shown in Table 1. Measurements above 4 pCi/L (the recommended maximum for permanent habitation) are frequent. As expected, basement levels are higher than main floor levels, but no difference is seen between main floor and main bedroom levels.

Table 2 shows the frequency distribution of radon daughter count rates from the subjects. While these counts were not performed under a completely standardized protocol with respect to time and previous activities, it is still worth noting that on a population basis approximately 79% of the subjects are above background (100% minus twice the percentage below zero) assuming a symmetrical distribution of counting errors. The fraction of subjects above 13.5 CPM ( $\pm 2$  standard deviations of the counting error) is 59%.

Table 3 shows the distribution of study subjects by sex and smoking category: females and nonsmokers predominate.

2023380561

Table 1.—Pennsylvania House Radon Levels for Residences of Measured Subjects

Radon quantiles	Radon levels (pCi/L) by house area		
	Basement	Main floor	Main bedroom
Quantile 25	1.9	1.0	0.9
50	3.2	1.7	1.6
75	6.2	2.8	2.8
90	17	5	5
95	31	14	14
100	49	52	31
Number of homes*	102	112	113
*Total houses represented: 124.			

Table 2.—Radon Daughter Contamination of Pennsylvania Residents\*

Counts per minute (CPM)†	Percentage of residents	
	In category	Cumulative
-17-0	10.6	10.6
0-12.4	25.0	35.6
12.5-24	27.7	63.3
25-49	25.6	88.9
55-99	6.1	95.0
100-199	2.8	97.8
200+	2.2	100

\*N = 180.  
†Two standard deviations of the background counting rate is 13.5. Counts are minus background.

Table 3.—Numbers of Subjects by Sex and Smoking Category with Available Main Floor Radon Measurements

	Male	Female	Total
Nonsmokers	30	56	86
Smokers	10	16	26
Total	40	72	112

Table 4 shows the statistical significance of the three variables of interest: household radon, sex, and tobacco smoking. As expected, household (main floor) radon is very highly significant and is the primary determinant of body contamination levels. In the arithmetic models, variables are more highly significant, especially with the three largest outliers excluded ( $r^2 = .65$ ).

Figure 1 shows graphically the wide spread of the data but also the clear negative association between

Table 4.—Regressions of Radon Daughters in/on Pennsylvania Residents

Regression models and terms	t	p
Basic model* ( $R^2 = .37$ )		
In Rn (pCi/L)	6.39	.001
Sex (F) x In Rn	2.37	.020
Smoker x In Rn	-2.88	.005
Extended model† ( $R^2 = .38$ )		
In Rn (pCi/L)		
Sex (F) x In Rn	2.35	.012
Smoker x In Rn	-2.71	.008

\*In CPM; a quadratic adjustment for travel time is also included.

†Model also includes a dummy position variable (p = .99) and a term weight x In Rn. The latter variable is so confounded with In Rn that neither are significant when Type II sums of squares are used.

current smoking and the presence of high radon daughter contamination levels.

Table 4 and Figure 1 relate body radon daughter contamination to radon levels in the main floor living area, because nearly all measurements were made in afternoon and evening hours. However, the multiple correlation using the logarithmic models was slightly higher if bedroom measurements were used ( $R^2 = .43$  vs.  $R^2 = .37$ ), while the correlation was essentially identical if basement levels were used ( $R^2 = .36$ ). This pattern holds also for the other models and when outliers are deleted.

Table 5 shows estimates of radon daughter contamination on subjects from both the arithmetic model (three outliers deleted) and the logarithmic model. Results are similar in the lower radon exposure ranges but diverge at higher radon levels, not unexpectedly as the logarithmic transformation reduces the weight of very high measurements. Without deletion of outliers, arithmetic estimates are yet higher, especially for the 10 male smokers.

Table 6 shows the ratios of the logarithmic estimates in Table 5. There appears to be a 2-3x excess of contamination in females at levels above 4 pCi/L, and a 2-4x excess among nonsmokers relative to smokers. The contamination of female nonsmokers relative to male smokers could be very high, up to sixteen-fold, according to the model.

The role of body size, which should be related to amount of contamination (and counting efficiency), was also investigated. Height (H), weight (W), and relative weight ( $W/H$ ,  $W/H^2$ ) were investigated. These variables are also related to both sex and cigarette smoking. In general, weight appears to be the most useful single variable, but statistical significance is variable because of the very high degree of confounding (of weight x Rn with Rn alone). Weight has been retained in the extended models, and estimates (Table 5) are presented for males and females of mean weight (166.5 lb):

2023380562

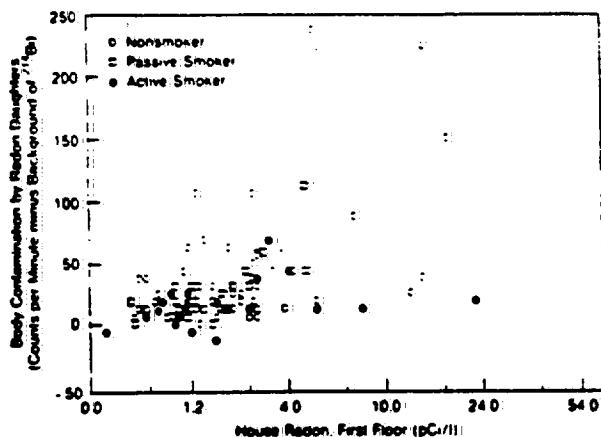


Fig. 1. Body radon daughter contamination vs. radon levels on first floor living area.

### Discussion:

The current study was adventitious, having been designed to study potential occupationally acquired body burdens of radium. Nevertheless it has some important implications. Most importantly, it demonstrates that body contamination with radon daughters is well predicted by radon levels measured on the first floor of the home over the 6-12 mo period following. While one would expect such a relationship, this direct demonstration is gratifying. The strength of the relationship is impressive given that the body contamination measurements were made at one point in time during the heating season. A series of such measurements would presumably yield a much higher correlation.

The equally high correlation of basement radon levels to personal contamination, relative to main floor levels, is of considerable relevance to epidemiological studies. When only short-term measurements can be taken, the basement is currently the measurement site of choice because radon levels are less variable in a closed basement than are first floor radon levels. That it will average higher than first floor levels is a drawback, but first floor exposures can be estimated statistically.

The finding that bedroom radon levels correlate

Table 6.—Ratios of Radon Daughters in/on Pennsylvania Residents at 1-Hr from Home: Extended Logarithmic Model.

First floor radon (pCi/L)	Nonsmoker/ smoker		Female/male:	
	M	F	Nonsmk.	Smk.
1	1.3	1.3	1.3	1.3
4	2.2	2.1	1.8	1.9
10	3.1	3.0	2.5	2.6
25	4.5	4.5	3.6	3.5

more highly with individual contamination even in afternoon and evening hours than do main floor living area measurements was unexpected. It does suggest that if a single long-term measurement is to be made, the bedroom may be the best choice of location.

In addition, the findings suggest that cigarette smoking is associated with lower levels of total body contamination. This does not directly support the relationship assumed in the current literature<sup>4-6</sup> that passive smoking increases lung dose by increasing the equilibrium ratio. The latter may occur, but there may be stronger countervailing factors, such as reduction of superficial plateout. Thoracic burdens need to be studied more directly.

Females are found to have higher body contamination levels than males at the same household levels; this may imply higher risks for females relative to males for domestic exposure. There is no necessary implication of intrinsic sex differences; that, for example, female miners would differ from male miners. Only heterogeneity of individual risk by sex, for a given household level of radon, is implied.

Although we consider this measurement of body contamination as an index of exposure levels in the breathing zone, the actual <sup>210</sup>Bi in/on an individual reflects four variables. These are, in probable order of magnitude: (a.) unsupported radon daughters contaminating clothes, hair, and skin; (b.) unsupported radon daughters inhaled and deposited in the upper and lower respiratory tract; (c.) supported radon daughters

Table 5.—Radon Daughters (CPM) in/on Pennsylvania Residents at 1-Hr from Home: Extended Arithmetic and Logarithmic Models

First floor radon (pCi/L)	Males				Females			
	Non-smokers		Smokers		Non-smokers		Smokers	
	Arith.	Log.	Arith.	Log.	Arith.	Log.	Arith.	Log.
1	15	12	9	9	24	16	18	12
4	34	26	9	12	68	48	43	23
10	72	49	9	16	157	124	93	41
25	167	98	8	22	379	348	220	77

<sup>†</sup>Three outliers deleted from regression.

2023280513

from radon in the body, especially in fatty tissue"; and (d.) unsupported radon daughters translocated from the respiratory tract.<sup>13</sup> Quantitative estimation of the relative importance of these four components or of their relationships to carcinogenesis is beyond the scope of this paper. A manuscript dealing with the more physiological aspects of this measurement technology is in preparation.

The problem of external contamination, external in the sense that it is on the outside of the subject, cannot be ignored. In this sense, the subject's breathing zone is also external, and the subject's hair, clothes, and skin can be thought of as a collector of radon daughters in that immediate environment. It seems unlikely that, over a very wide range of exposures, persons, and homes, internal and external burdens would not correlate highly. And that is the question: do they correlate highly? Their relative magnitudes are in this context of much less importance.

Preliminary experimental results on five male subjects in our laboratory suggest a high correlation of internal ("nonremovable") and external ("removable") radon daughter contamination: over a narrow two-fold range of observed counts (a narrow range will reduce the correlation), the correlation coefficient between "removable" and "nonremovable" daughters is .90 ( $p < .05$ ).

The general implications of the findings reported here do not necessarily relate only to physical-environmental factors, but also to physical-personal factors and to biological or social variables.

Physical characteristics of persons might relate to deposition rates on body surfaces or clothing. Women may wear more synthetic fabrics, which generate a high static charge enhancing deposition. Conversely, cigarette smoke might neutralize or reverse charge characteristics of body surfaces, or otherwise modify deposition characteristics. Electrical characteristics of upper respiratory surfaces may partially determine the sites of deposition of very small electrically charged air ions, including radon daughters.<sup>14</sup>

Roles of biological characteristics (other than surface area and respiratory minute volume) are more obscure. As noted above, sex differences in effective body thickness were minimal (3%). Pulmonary diffusion capacity for radon would be expected to be impaired in smokers, but such an effect might be so minor as not to be measurable. Body fat content is lower in both smokers and males, and this would lower the body pool of radon.

Social and psychological factors are likely, we believe, to predominate. Women are more likely than men to be at home all day. Even with respect to cigarette smoking, it might be hypothesized that the smoker is more likely to be active, shopping, visiting, etc. Such factors relating to personal time utilization are likely both to be very important and to relate directly to lung cancer risk from radon daughters.

In current risk assessment for radon, it is generally assumed that the exposure term from surveys of houses, dose estimates from current models, and dose-response estimates from published studies of miners

are all that are required to perform a useful health risk assessment relating to radon for the general population. This may prove to be an oversimplification.

Analyses of these existing data are being continued in several directions: the use of detailed positional information on contamination, the possible use of the full spectrum to maximize available information, and analyses of data on activities of subjects earlier on the day of measurement.

\*\*\*\*\*

Data were collected under U.S. Department of Energy Contract W-31-109-ENG-38. Statistical analyses were carried out under NIH Grant 5 R01 CA 40071. The contributions of G. A. Anast, M. A. Edling, T. A. Harper, D. R. Kardatske, T. J. Kotek, A. Pagnamenta and R. E. Toohey are gratefully acknowledged.

Requests for reprints should be sent to: James H. Stebbings, Sc.D., Bldg. 202, Biological Environmental and Medical Research Division, Argonne National Laboratory, Argonne, IL 60439-4833.

\*\*\*\*\*

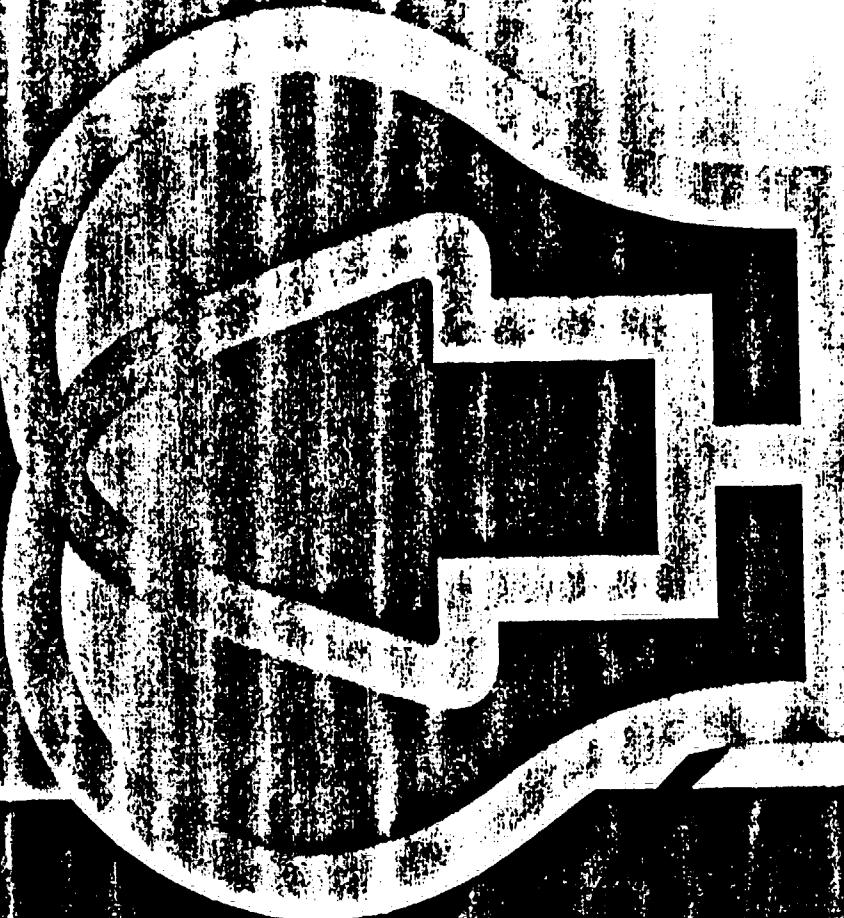
#### References

1. Nero, A. V.; Schwehr, M. B.; Nazaroff, W. W.; and Revzan, K. L. 1986. Distribution of airborne radon-222 concentrations in U.S. homes. *Science* 234:992-97.
2. Alter, H. W. and Ottavald, R. A. 1987. Nationwide distribution of indoor radon measurements: A preliminary data base. *JAPCA* 37:227-31.
3. National Council on Radiation Protection and Measurement. 1984. Exposures from the Uranium Series with Emphasis on Radon and its Daughters. NCRP Report No. 77, Bethesda, MD.
4. Lebowitz, M. D. 1986. The potential association of lung cancer with passive smoking. *Environ Int* 12:3-9.
5. National Council on Radiation Protection and Measurement. 1984. Evaluation of Occupational and Environmental Exposures to Radon and Radon Daughters in the United States. NCRP Report No. 78, Bethesda, MD.
6. Winters, T. H. and DiFrenza, J. 1983. Radioactivity and lung cancer in active and passive smokers. *Chest* 84:653-54.
7. Bergman, H. and Axelson, D. 1983. Passive smoking and indoor radon daughter concentrations. *Lancet* 2:1308-09.
8. Bergman, H.; Edling, C.; and Axelson, D. 1984. Indoor radon daughter concentrations and passive smoking. In: Proceedings of the 3rd International Conference on Indoor Air Quality and Climate, Stockholm, August 20-24. B. Berglund, T. Lindvall, J. Sundell, Eds., Vol. 2, pp. 79-84. Stockholm: Swedish Council for Building Research.
9. Ginevan, M. E. and Mills, W. A. 1986. Assessing the risks of Rn exposure: The influence of cigarette smoking. *Health Phys* 51:163-74.
10. Toohey, R. E.; Keane, A. T.; and Rundo, J. 1983. Measurement techniques for radium and the actinides in man at the Center for Human Radiobiology. *Health Phys* 44 (Suppl 1): 323-42.
11. Rundo, J.; Sha, J. Y.; and Toohey, R. E. 1979. Radon daughters in the occupants of a house with an unusually high concentration of airborne radon. In: Radiological and Environmental Research Division Annual Report, Report ANL-79-65, pp. 13-20. Argonne, IL: Argonne National Laboratory.
12. SAS Institute. 1982. User's Guide: Statistics. Cary, NC: SAS Institute.
13. Pohl, E. 1964. Dose distribution received on inhalation of  $Rn^{222}$  and its decay products. In: Radiological Health and Safety in Mining and Milling of Nuclear Materials, Vol. I, pp. 221-236. Vienna: International Atomic Energy Agency.
14. Minnesota Environmental Quality Board. Science Advisors 1982. A Health and Safety Evaluation of the 400 KV DC Powerline, pp. III-6, III-11. St. Paul, MN: Minnesota Environmental Quality Board.

2023380564

2023380565

# INDOOR AIR '90



THE FIFTH  
INTERNATIONAL  
CONFERENCE  
ON INDOOR AIR  
QUALITY AND  
CLIMATE

## THE INFLUENCE OF ENVIRONMENTAL TOBACCO SMOKE ON RADON DOSIMETRY

J.N. Pritchard and J.C. Strong  
Biomedical Research Department,  
AEA Environment and Energy,  
Building 551, Harwell Laboratory,  
Oxfordshire, OX11 ORA,  
England.

Environmental tobacco smoke (ETS) can affect the radiation dosimetry of indoor radon by modifying the behaviour of its decay products. Firstly, ETS significantly enhances the concentration of airborne particles, thereby reducing the unattached fraction. However, the lower mobility of ETS particles leads to an increase in airborne radioactivity by a factor of 2.5. Calculations presented here suggest that this is likely to give a protective effect, although an increased dose can result, depending upon how low the unattached fraction is in the absence of ETS. Clearly, this merits further study. Secondly, natural radioactivity in tobacco means that ETS enhances the airborne concentrations of the long-lived decay products  $^{210}\text{Pb}$  and  $^{210}\text{Po}$  by a factor of up to 2.4 for the latter nuclide. However, this does not significantly increase the overall dose due to radon. Finally, it has been suggested that the act of smoking can enhance the deposition of the attached fraction in the bronchial region; this hypothesis has been discounted.

## **INTRODUCTION**

Radon is the single most important source of irradiation of the general population (1). Most of this dose derives from the deposition in the respiratory tract of the short-lived decay products of radon ( $^{218}\text{Po}$ ,  $^{214}\text{Pb}$ ,  $^{214}\text{Bi}$  and  $^{214}\text{Po}$ ), two of which are alpha-emitters (2). When radon decays, the products are present as free ions, typically 3 nm in size (3). These have a high mobility, due to Brownian diffusion, with the result that they deposit to a surface, be it the fabric of a room, an ambient airborne particle or the surface of the respiratory tract. The airborne fraction present on ambient particles, which have an average size of about 130 nm (3), is termed 'attached', the remainder being 'unattached'. The unattached fraction deposits more efficiently in the respiratory tract than the attached; the site of deposition is also shifted from the pulmonary (attached) to bronchial (unattached) regions of the lung (4). The International Commission on Radiological Protection (ICRP) have recently calculated that the dose to the respiratory tract is dominated by that to the bronchial region, with that due to the unattached fraction being at least an order of magnitude greater than that due to the attached (4). Environmental tobacco smoke (ETS) can affect dosimetric calculations of indoor radon decay products in a number of ways. Firstly, cigarette smoking significantly enhances the concentration of

Source: <https://www.industrydocuments.ucsf.edu/docs/hnx0000>

airborne particles. This will have the effect of reducing the unattached fraction, thereby reducing the resulting radiological dose. However, the mobility of such particles is much less than for the unattached fraction, with the result that more of the decay products remain airborne for longer periods. Thus, the presence of ETS may either reduce or enhance the dose, depending on the prevailing conditions in the absence of ETS. Secondly, it is well documented that tobacco contains the two long-lived radon decay products,  $^{210}\text{Pb}$  and  $^{210}\text{Po}$ , the latter being an alpha-emitter (5). Both are relatively volatile metals, and so may significantly increase the ambient levels of these nuclides during tobacco combustion. Lastly, it is known that the deposition of tobacco tar during the act of smoking does not follow predictions based on particle size (6). Should this also apply to ETS, or if a substantial fraction of decay products be inhaled during the act of smoking, a revision in dosimetric estimates is required (7). This paper considers each effect in turn.

#### EFFECTS OF ETS ON SHORT--LIVED DECAY PRODUCTS

ETS significantly increases the number of airborne particles present in indoor atmospheres. For example, a cigarette contributes about 10 mg of particles of median size 200 nm in ETS (8); assuming such particles are unit density spheres, the concentration in an unventilated 14 m<sup>3</sup> room would be  $9 \cdot 10^4$  cm<sup>-3</sup>, well above normal ambient levels. Clearly, such an increase in particle concentration will reduce the unattached fraction. Normally, 7-15 % of the decay products are unattached, but this drops to less than 5 % in the presence of a source of airborne particles such as ETS (9). However, such attachment means that the decay products will remain airborne longer, since the mobility of the particles due to Brownian diffusion will be much less than that of the free ions. This, in turn, will lead to an increase in the levels of total airborne radioactivity; under experimental conditions, it has been demonstrated that the smoking of cigarettes leads to an increase in the concentration of decay products by a factor of 2.5 (10).

The current ICRP lung model (11) does not apply to radionuclides with short half-lives, resulting in the critical dose to the bronchial region being underestimated (4). This situation is -being rectified (4); following unit exposure, calculated doses to the bronchial region are 150 nGy for 3 nm particles (unattached), 11 nGy for 130 nm particles (ambient attached) and 7 nGy for 200 nm particles (ETS). [Unit exposure is defined as 1 Bq h m<sup>-3</sup> EER; EER is the equilibrium equivalent concentration of radon, i.e. the activity concentration of radon in equilibrium with its decay products which has the same potential alpha-particle energy as the actual non-equilibrium mixture of decay products.] Thus, for an ambient atmosphere of 130 nm particles with an unattached particle size of 3 nm, and fraction (F), the dose per unit exposure (in nGy per Bq h m<sup>-3</sup>) is:-

$$D = 150F + 11(1-F)$$

$$= 139F + 11 \quad (1)$$

2023380567

In the presence of ETS particles of 200 nm, this equation becomes:-

$$D = 143F + 7$$

(2)

Thus, the presence of ETS reduces the overall dose for a given exposure and degree of attachment. Against this, it should be noted that the presence of cigarette smoke enhances the airborne concentration by a factor of 2.5, with the result that exposures will be 2.5 times greater (10), thereby increasing the dose for a given degree of attachment. However, the degree of attachment is a strong function of the airborne particle concentration (12).

It has been found from a study of German homes, that the particle concentration could increase 100-fold during the act of cigarette smoking, remaining elevated for up to 5 hours afterwards (9). Correlating with this, the average unattached fraction dropped from an average of 0.12 to 0.02 (9), whilst the average radon concentrations increased from 160 Bq m<sup>-3</sup> EER to 295 Bq m<sup>-3</sup> EER. Thus, from equation (1), one hour's exposure would lead to a dose of 4.4  $\mu$ Gy prior to smoking, but only 2.9  $\mu$ Gy after smoking (equation (2)), a drop of 34 %. Making the simplified assumptions that smoking always reduces the unattached fraction to 1/6 of its previous value (9), but that the overall concentration of radioactivity rises by a factor of 2.5 (10), then the presence of cigarette smoke will lead to a dose reduction, when the original unattached fraction is greater than a value, F, found by solving the equation:-

$$139F + 11 = (143F/6 + 7) \times 2.5$$

(3)

i.e. for values of the unattached fraction prior to smoking of greater than 8.2 %. This value is well below the average 18 % observed in UK dwellings (3), and below the average 10 % in German houses without obvious aerosol sources such as smoking or cooking (9).

In view of these calculations, it is perhaps surprising that there have not been more investigations of the effects of the indoor aerosol on the attached fraction and, hence, dose. Some limited evidence to support these calculations comes from a comparative study of the levels of <sup>214</sup>Bi in smokers and nonsmokers, determined using whole-body monitoring (13). Non-smokers were found to have 2-4 times the levels of smokers. Unfortunately, the data were not described in terms of whether the nonsmokers lived with another non-smoker, or a smoker. However, given that smokers must live in an atmosphere where ETS is present, these data would suggest that the dose from the shortlived decay products is indeed lower.

#### EFFECTS OF ETS ON LONG-LIVED DECAY PRODUCTS

Tobacco, as any other plant, has a tendency to incorporate radio-nuclides in the environment. Two nuclides of particular dosimetric significance are <sup>210</sup>Pb and <sup>210</sup>Po (14). Both are relatively volatile

2023380568

metals, with the result that a significant proportion of the cigarette content may become airborne during combustion, thereby raising the concentration of these nuclides. Certainly, it is well documented (e.g. 15) that cigarette smokers have higher burdens of these nuclides as a result of their intake through smoking.

On average, cigarette tobacco contains 17 mBq g<sup>-1</sup> of 210Po; this is supported by an equal activity of the parent nuclide (5). Estimates suggest that between 24 and 46 % of the cigarette 210Po is transferred to ETS (16,17,18); 210Pb has about half the volatility of 210Po (19,20). Assuming that a cigarette contains 0.8 g of tobacco, there will be approximately 4.8 mBq of 210Po and 2.4 mBq of 210Pb generated in ETS. Thus, if 20 cigarettes are smoked over the course of 16 hours in a 14 m<sup>3</sup> room, being ventilated at 3 air changes per hour, then average levels generated of 210Po and 210Pb will be 0.14 and 0.07 mBq m<sup>-3</sup> respectively.

To put these values into context, the average 210Pb concentration in air is estimated to be 0.5 mBq m<sup>-3</sup> (21). As the ratio of 210Po to 210Pb is about 0.2 (1), the concentration of 210Po is 0.1 mBq m<sup>-3</sup>. Thus, the addition of ETS will only increase the levels of 210Pb by 14 %, but those of 210Po by 140 %, to 0.24 mBq m<sup>-3</sup>. Since the latter nuclide is an alpha-emitter, it dominates the dose arising from inhalation of these two nuclides, contributing 86 % of the total (1). Using the current ICRP model (11), and assuming 0.2 µm ETS particles with pulmonary clearance half-times of half with 0.2 d and half with 0.01 d (Class W), the committed effective dose equivalent for 210Po is 3.8 µSv Bq<sup>-1</sup>. Further assuming a breathing pattern of 0.75 m<sup>3</sup> hr<sup>-1</sup>, then the total intake per year is 1.6 Bq, i.e. a dose of 6 µSv per year. Under the proposed new model, the clearance rates are slightly faster (22) and the deposition a factor of 3 lower (4), so the calculated dose will be much smaller. Since the total dose due to the inhalation of radon and decay products is about 1000 µSv per year (1), it can be seen that the contribution from natural radioactivity in ETS is insignificant.

#### THE DEPOSITION OF TOBACCO SMOKE

The above calculations assume that the current and proposed deposition models apply to tobacco smoke. Many factors are known to influence deposition other than size and breathing pattern, including physique, disease and particle solubility (23). In particular, the deposition of tar from cigarette smoke during the act of smoking is much greater, and more central within the lung, than would have been predicted from its particle size; in fact, it deposits like a particle some 10 times larger (6). However, the limited data on the deposition of ETS would suggest that the processes of aging and dilution change the nature of the smoke, resulting in a deposition pattern like any other non-hygroscopic 200 nm particle (24).

Finally, it has been suggested that the particles of ambient air, whether ETS or not, could be drawn through a cigarette during the act of puffing. This could modify their deposition pattern as outlined above, thereby contributing to 'hot' spots of activity in the lung (7). However, this hypothesis has been discounted, since

2023380569

Limits for intakes of radionuclides by workers. ICRP Publication 30, Part 1. Ann ICRP 2(314)-

12. Duggan MJ and Howell DM (1969) Relationship between the unattached fraction of airborne RaA and the concentration of condensation nuclei. Nature 224:1190-1191.
13. Stebbings JH, Kardatzke DR, Toohey RE, Essling MA and Pagnamenta A (1986) Domestic and personal determinants of the contamination of individuals by household radon daughters. Trace Subst Environ Health 20:392-400.
14. Watson AP (1985) Polonium-210 and lead-210 in food and tobacco products: transfer parameters and normal exposure and dose. Nucl Safety 26:179-191.
15. Cohen BS, Eisenbud M, Wrenn ME and Harley NH (1979) Distribution of polonium-210 in lung. Radiat Res 79:162-168.
16. Ferri ES and Baratta EJ (1966) Polonium-210 in tobacco products and human tissues. Radiol Health Data Rep 7:485-488.
17. Black SC and Bretthauer EW (1968) Polonium-210 in tobacco. Radiol Health Data Rep 9:145-152.
18. Harley NH, Cohen BS and Tso TC (1978) Radioactivity in Consumer Products (Moghissi AA, Paras P, Carter MW, Barker RF, eds). Nuclear Regulatory Commission, Washington DC. p.199-216.
19. Ferri ES and Christiansen H (1967) Lead-210 in tobacco and cigarette smoke. Public Health Rep 82:828-832.
20. Cohen BS, Eisenbud M and Harley NH (1980) Alpha radioactivity in cigarette smoke. Radiat Res 83:190-196.
21. Jaworski Z (1969) Radioactive lead in the environment and in the human body. Atomic Energy Rev 7:3-45.
22. Johnson JR and Milencoff S (1989) A comparison of excretion and retention between the current ICRP model and a proposed new model. Health Phys 57 (Suppl 1):263-270.
23. Evans JC, Pritchard JN, Black A, Hicks JF and Morgan JR (1987) Deposition and clearance of aerosols in the human respiratory tract (Hofmann W, ed). Facultas Universitatisverlag, Vienna. p.63-68.
24. Hiller FC, Mazunder MK, Wilson, JD, McLeod PC and Bone RC (1982) Human respiratory tract deposition using multi-modal aerosols. J Aerosol Sci 13:337-343.
25. Cross PT (1984) Radioactivity in cigarette smoke issue. Health Phys 46:205-208.

2023380570

Limits for intakes of radionuclides by workers. ICRP Publication 30, Part 1. Ann ICRP 2(314)-

12. Duggan MJ and Howell DM (1969) Relationship between the unattached fraction of airborne RaA and the concentration of condensation nuclei. Nature 224:1190-1191.

13. Stubbings JH, Kardatzke DR, Toohey RE, Essling MA and Pagnamenta A (1986) Domestic and personal determinants of the contamination of individuals by household radon daughters. Trace Subst Environ Health 20:392-400.

14. Watson AP (1985) Polonium-210 and lead-210 in food and tobacco products: transfer parameters and normal exposure and dose. Nucl Safety 26:179-191.

15. Cohen BS, Eisenbud M, Wrenn ME and Harley NH (1979) Distribution of polonium-210 in lung. Radiat Res 79:162-168.

16. Ferri ES and Baratta EJ (1966) Polonium-210 in tobacco products and human tissues. Radiol Health Data Rep 7:485-488.

17. Black SC and Bretthauer EW (1968) Polonium-210 in tobacco. Radiol Health Data Rep 9:145-152.

18. Harley NH, Cohen BS and Tso TC (1978) Radioactivity in Consumer Products (Moghissi AA, Paras P, Carter MW, Barker RF, eds). Nuclear Regulatory Commission, Washington DC. p.199-216.

19. Ferri ES and Christiansen H (1967) Lead-210 in tobacco and cigarette smoke. Public Health Rep 82:828-832.

20. Cohen BS, Eisenbud M and Harley NH (1980) Alpha radioactivity in cigarette smoke. Radiat Res 83:190-196.

21. Jaworowski Z (1969) Radioactive lead in the environment and in the human body. Atomic Energy Rev 7:3-45.

22. Johnson JR and Milencoff S (1989) A comparison of excretion and retention between the current ICRP model and a proposed new model. Health Phys 57 (Suppl 1):263-270.

23. Evans JC, Pritchard JN, Black A, Hicks JP and Morgan JR (1987) Deposition and clearance of aerosols in the human respiratory tract (Hofmann W, ed). Facultas Universitatsverlag, Vienna. p.63-68.

24. Hiller FC, Mazunder MK, Wilson, JD, McLeod PC and Bone RC (1982) Human respiratory tract deposition using multi-modal aerosols. J Aerosol Sci 13:337-343.

25. Cross FT (1984) Radioactivity in cigarette smoke issue. Health Phys 46:205-208.

2023380571

# RISK ASSESSMENT OF EXPOSURE TO INDOOR AIR POLLUTANTS

J. J. McAughey, J. N. Pritchard and A. Black

61 [① ~~DAY~~ Environmental and Medical Sciences Division  
② Harwell Laboratory, Didcot, Oxon OX11 0RA, UK  
③ (OXON ENGLAND)]  
(Received 29 January 1990; accepted 8 March 1990)

## ABSTRACT

Risk assessment of exposure to indoor air pollutants should be a consideration not simply of airborne concentration, but of individual or population dose. Dose, the uptake and retention of the substance, may be affected by the physical and chemical form of the pollutant, its local environment, and physiological factors. Facilities for controlled exposures to quantify these effects in animals and humans will be described with particular reference to the dosimetry of radon gas and its progeny, and environmental tobacco smoke. The use of radioactive and stable isotope tracers in such studies will also be discussed.

## INTRODUCTION

Many factors may affect the human uptake and retention of specific indoor air pollutants, both at work and in the home. These include the physical form of the pollutant, that is, whether it is in the gaseous, vapour or particulate phase, and the shape, size and solubility of particles. Similarly, for particle deposition, the environment in which exposure occurs is important with respect to factors such as temperature, humidity and ventilation. Finally, physiological factors such as physique, disease and inter-subject variability may affect regional particle deposition and hence, dosimetry. Therefore it is important that controlled volunteer and animal exposures be carried out to determine the relative importance of these factors in population exposure to specific pollutants. Volunteer exposures may be used to determine deposition, uptake, metabolism and excretion of pollutants with animal exposures yielding additional information relating dose and metabolism to biological effects. It is only by considering all these factors that valid models of risk estimate may be obtained.

## FACTORS AFFECTING DOSIMETRY

### Gas and vapour deposition

The uptake and retention of vapours and gases is fairly well understood and is dependent primarily on the chemical nature of the gas or vapour and its interaction with the surface of the respiratory tract. In each case there is rapid mixing within the airways and fast diffusion to the lung surface. For vapours, high levels of deposition are likely to be seen for soluble compounds and those which chemically 'fix' to the lung surface. The principles governing these processes have been reviewed by Davies (1). However, it should be noted

20230821  
COPYRIGHT  
Selper Ltd., FUELIC  
TICKS DIV. LONDON ENGLAND

that lipophilic vapours may also be taken up through the skin and may make a significant contribution to the absorbed dose.

#### Particle deposition

It is important to consider the mechanisms of particle deposition and clearance in the respiratory tract such that changes in total and regional deposition through external factors might be predicted and taken into account when risk assessments are carried out. The most important mechanisms are:-

- a) Impaction, due to the inertia of the particles when the air undergoes a change of direction, such as in the pharynx or at a bifurcation. This is important for the deposition of larger particles in the upper airways where fast mucociliary clearance occurs.
- b) Sedimentation, due to the action of gravity.
- c) Brownian motion, due to the bombardment of the particles by air molecules. This is most important for the deposition of smaller particles in the alveolar region. This region has a high surface area allowing rapid dissolution and uptake of soluble particles. For insoluble particles macrophage clearance predominates. The rate of clearance of lipid soluble compounds tends to be related to their molecular weight, with more rapid clearance at mass less than 300 Da (2).

The deposition of particles is more affected by external factors than gas or vapour deposition. Data linking particle size to regional deposition in the lung has been published by the International Committee on Radiological Protection (3) for the naso-pharyngeal region (NP), the tracheo-bronchial region (TB) and the alveolar or pulmonary region (P) of the airways. However, to date, most of the available deposition data comes from a relatively small number of healthy male Caucasian subjects who have inhaled spherical insoluble particles. When assessing risk from airborne pollutants it is possible that none of these conditions may apply.

#### Particulate and environmental factors

As stated already, particle size is an important determinant of regional deposition but shape and solubility of the inhaled particle also have an important role. For example, fibrous materials such as asbestos are known to be injurious to the lung. When airborne, the aspect ratio of the fibres (length/diameter) may reach 1000, very different from the spheres commonly studied. However, the fibres tend to align with the airflow so that the aerodynamic diameter is not greatly dependent on length. Thus fibres up to 360  $\mu\text{m}$  in length have been found to penetrate to the human alveoli (4). With fibres of this length it is impossible for normal alveolar clearance to operate and the defence mechanism is to form proteinaceous bodies around the fibres in an effort to render them harmless.

On entering the high humidity of the respiratory tract, water vapour will condense on a particle. If soluble, the particle hydrates, eventually forming a solution. Since the vapour pressure of a solution is less than that of the pure solvent, the particle may swell to a droplet several times its initial size, known as hygroscopic growth. In the case of sodium chloride, growth of a factor of six is observed, which implies a marked shift in the deposition pattern of sub-micron particles (5). In practical terms, this is of considerable importance. The majority of the ambient aerosol is hygroscopic (6) due to acid pollutants together with sodium chloride in marine environments. Thus, potential environmental hazards, such as radon, may become

2023380573

attached to the ambient aerosol either by condensation or coagulation, and deposit as a hygroscopic material.

#### Physiological factors

The shape and size of an individual's airways will have an effect on the efficiency of deposition. Alveolar dimensions are unlikely to vary significantly, as alveolar diameter has been shown only to vary with body weight to the power 0.2 (7). However, bronchial dimensions are more strongly dependent on physique; on average, the female trachea is 1.3 times smaller than the male (8). Since the flow of air and hence the momentum of any particle is inversely proportional to the square of the airway diameter, deposition by impaction in the upper airways shows clear differences between men and women because impaction efficiency increases at a given flow (9).

In addition, lung disease may lead to regions which are poorly ventilated, possibly accompanied by airway obstruction. This will lead to changes in airflow patterns and hence deposition. Similarly, mucus hypersecretion may affect airway diameter and the rate of removal of deposited particles, thus altering dose.

These factors combined give rise to substantial inter-subject variability of deposition in different regions within the respiratory tract. For example, in a series of four experiments described by Hicks et al (10) the proportion of the lung deposit in the TB region showed consistent differences between five subjects ranging from 40-80%. Thus ranges of deposition ought to be considered in each case rather than single line deposition versus size models.

#### Population dosimetry

As noted above, many factors may contribute to a range of dose estimates through uptake and retention. However, for population risk estimates further factors may have to be taken into account, particularly with respect to differences in metabolism and excretion, and ultimately, biological effect. Thus, certain groups of the population may be more susceptible to particular pollutants through differences in biochemistry, induced either through environmental factors such as nutritional status, or genetically, by inheritance.

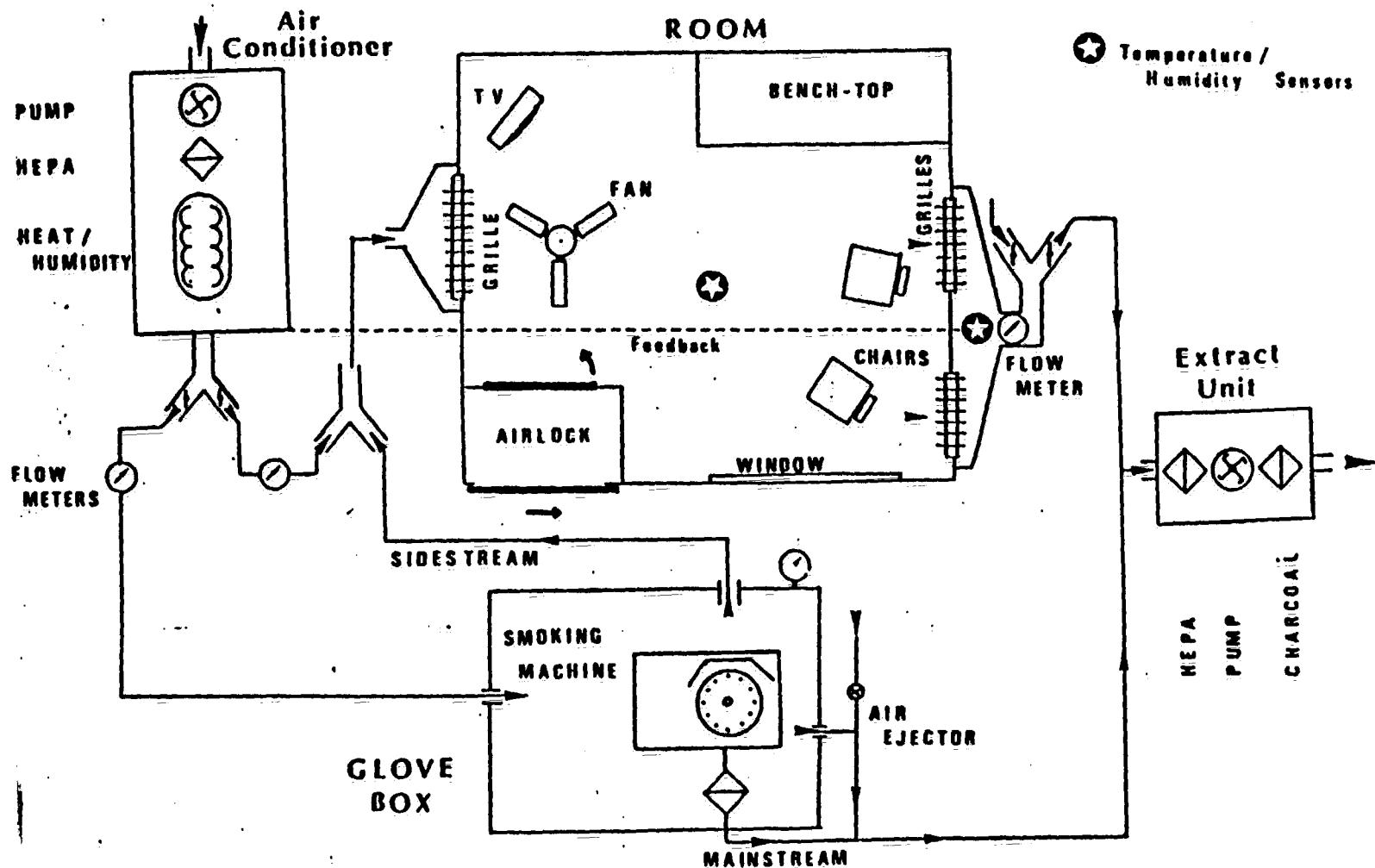
## EXPOSURE FACILITIES

#### Animal exposure facilities

Animal exposures may be divided into those where the whole body is exposed and the animals are generally unrestrained, or nose-only systems where restraining tubes may be used for rodents or mask systems for higher mammals, although both types of system have disadvantages. In restraining systems the animal may be under some degree of stress, thus affecting its breathing pattern and hence deposition. However, in whole body exposure systems, although animal behaviour may be more natural, it may become difficult to apportion the dose between that inhaled and that which is subsequently ingested from cleaning of the skin. At the Harwell Laboratory, both types of facility exist and have been used for studies of radio-nuclide and cigarette smoke dosimetry (11,12). More recently, a new pair of  $3.4 \text{ m}^3$  whole body exposure chambers have been built to allow studies of radon dosimetry from both short and long term exposures. An associated air handling plant has also been developed to allow continuous exposures for periods up to 3 months. In addition, this system will allow the investigation of potential synergy effects between radon and various vector aerosols. Of these, environmental tobacco smoke, and formaldehyde from cavity wall insulation are of significance for indoor air.

248

Figure 1: Exposure system for sidestream smoke atmospheres



2023380375

299

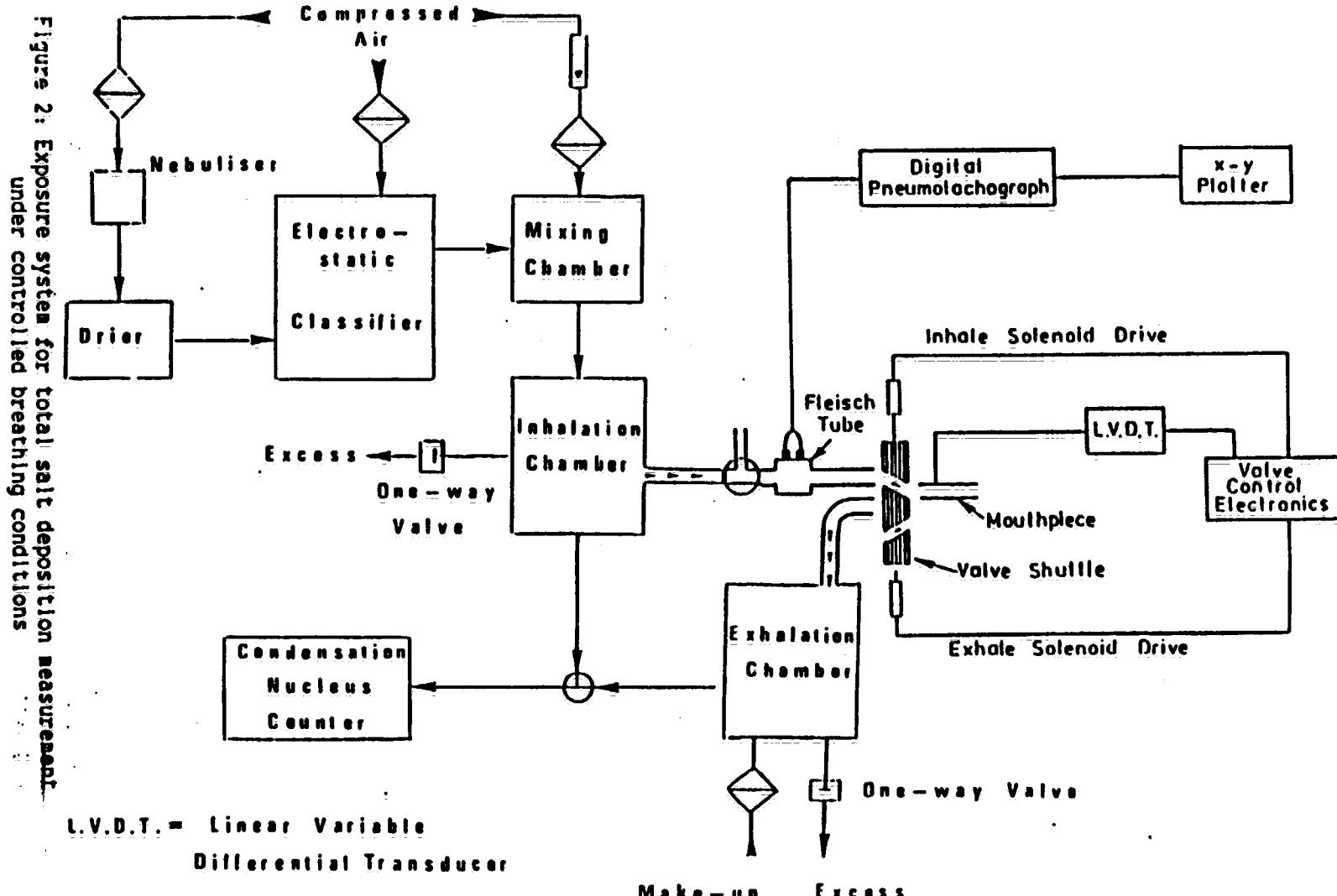


Figure 2: Exposure system for total salt deposition measurement under controlled breathing conditions.

2023380376

### Human exposure facilities

As for animals, volunteer exposures may be carried out in different ways, including whole body exposure, mouth-only or oro-nasal breathing. At the Harwell Laboratory, a series of facilities have been built for mouth-only and whole body exposures, particularly with respect to cigarette smoking and exposure to environmental tobacco smoke. The whole body system, a 14 m<sup>3</sup> chamber with associated air handling facilities has been described in greater detail elsewhere (13), and has also been used to investigate the physical behaviour of sidestream cigarette smoke as it ages and is diluted (14). A schematic of this chamber is shown in Figure 1. The mouth-only exposure apparatus is shown in Figure 2, as configured for studies of the effect of hygroscopic particle growth on total and regional deposition under controlled breathing patterns (15). This allows the effect of initial particle size, flow rate, volume and breath holding on deposition to be quantified.

### TRACER COMPOUNDS

Tracer compounds can be of great use in controlled exposures when assessing risk, either as analogues of behaviour, or to distinguish the administration of an experimental dose from continuing environmental exposure. Examples of radiotracers which have been used in work carried out by this laboratory include the use of <sup>123</sup>I-1-iodohexadecane as a marker for the particulate phase of both mainstream and sidestream cigarette smoke after extensive characterisation work (16). The use of this compound has given information on the physical behaviour of the smoke, its equilibrium between vapour and particulate phases, and its deposition and clearance in both animal (12) and volunteer studies (17). Other work has been carried out using <sup>38</sup>Cl labelled chlorofluorocarbons, which followed the fate of a series of compounds following inhalation in volunteers, and found their retention in the body to be related to the *in vitro* blood-air partition coefficient, thus allowing estimates for retention to be made for related compounds, where labelling was impractical (18). This stresses the importance of experimental work of this type, allowing input into risk models.

More recently, new powerful analytical techniques such as nuclear magnetic resonance (NMR), magnetic resonance imaging (MRI) and inductively coupled plasma - mass spectrometry (ICP-MS) have opened up the potential for stable isotope tracer studies without radioactivity, allowing wider population studies in vulnerable groups. For example, studies of the source apportionment of lead intake by inhalation from petrol additives versus other sources of lead, such as diet, as reviewed by Chamberlain (19) is typical of the potential of these techniques. This suggested airborne lead to be responsible for approximately 10% of lead uptake.

### APPLICATIONS

#### Environmental tobacco smoke dosimetry

The uptake and retention of environmental tobacco smoke (ETS) relative to the retention of active smokers serves as a useful example. Two factors are of particular importance: the aerodynamic particle size of the smoke aerosol as seen by the lung, and the status of the vapour-particle equilibrium of the aged ETS. In effect, directly inhaled tobacco smoke is subject to growth by coagulation and hygroscopicity from the original aerosol of 0.8 µm AMAD. In contrast, sidestream smoke has aged and been diluted prior to inhalation and is typically insoluble and of 0.2 µm AMAD. In addition, up to 70 per cent of the initial mass of the sidestream particulate has evaporated (14) such that the principles governing vapour deposition will become of greater significance.

22080521  
2023380521

Thus, if calculations of tracheo-bronchial particulate deposition are carried out, a relative risk ratio of 0.00017 between active and passive smokers is found (17) which is significantly lower than the dose estimates expected from reported epidemiological data if a comparable hazard exists.

#### Radon dosimetry

Radon is a naturally occurring radioactive gas, the dosimetry of which can vary markedly, depending on the form of radon and its progeny when inhaled. As radon gas, any material inhaled is immediately exhaled and hence deposition is low. Radon progeny, in contrast, are effectively metal atoms which will diffuse very rapidly to attach on airborne particles, room surfaces or within the TB region of the respiratory tract increasing radiation dose to the lung. In the presence of ETS, two conflicting mechanisms are likely to occur affecting dosimetry. Initially, due to increased airborne particle concentrations, the radon progeny are more likely to attach to the ambient particulate rather than surfaces and hence the airborne concentration of radioactivity is likely to increase. However the particles that stay airborne longest (approximately 0.5  $\mu\text{m}$ ) coincide with the size giving minimum total lung deposition. Thus, attachment of the radon progeny to the ambient particulate may give a protective effect with respect to radiation dose. It should be noted, however, that if the ambient aerosol is principally hygroscopic, this will again increase TB deposition and hence, radiation dose. Therefore, any estimates of risk must take into account all of these factors.

#### CONCLUSIONS

From the examples given above for ETS and radon dosimetry it can be seen that a combination of many factors have to be considered when calculating the uptake and retention of various pollutants, and that airborne concentration information is simply insufficient to properly develop population risk estimates. Added to this, individual differences in physique, breathing pattern, deposition, clearance and metabolism may also occur. These factors and differences in susceptibility to biological effect all have to be taken into account to assess individual and population risk.

#### BIBLIOGRAPHY

1. C.N. Davies, Ann. Occup. Hyg., 29, 13-25 (1985)
2. R.F. Henderson, W.E. Bechtold, M.A. Medinsky, J.P. Fischer and T.T. Lee, Toxicol. Appl. Pharmacol., 95, 515-521 (1988)
3. International Committee on Radiological Protection, Publication 26, Recommendations of the ICRP, Pergamon Press, Oxford (1966)
4. A. Morgan and A. Holmes, Brit. J. Ind. Med., 37, 25-32 (1980)
5. G.B. Xu and C.P. Yu, Aerosol Sci. Technol., 4, 455-461 (1985)
6. J.F. Hicks and W.J. Megaw, J. Aerosol Sci., 16, 521-527 (1985)
7. G.B. Xu and C.P. Yu, Aerosol Sci. Technol., 7, 117-123 (1987)
8. R. Warwick and P. Williams, eds., in Gray's Anatomy (34th Ed.) Longmans, London (1973).

2023380578

9. J.N. Pritchard, S.J. Jeffries and A. Black, J. Aerosol Sci., 17, 385-389 (1986)
10. J.F. Hicks, J.N. Pritchard, A. Black and W.J. Megaw in Aerosols: Formation and Reactivity, W. Schikarski, H.J. Fissan and S.K. Friedlander, eds., Pergamon Press, Oxford (1986)
11. M. Walsh, J.N. Pritchard, A. Black, S.R. Moores and A. Morgan, J. Aerosol Sci., 11, 467-474 (1980)
12. J.N. Pritchard, J.J. McAughey and A. Black, J. Aerosol Sci., 19, 1117-1120 (1988)
13. A. Black, J.N. Pritchard and M. Walsh, J. Aerosol Sci., 18, 757-760 (1987)
14. J.N. Pritchard, A. Black and J.J. McAughey, in Indoor Air Quality, R. Perry and P.W. Kirk, eds., Selper, London p. 49 (1988)
15. J.N. Pritchard, in Aerosols: Their Generation Behaviour and Application. Proceedings of the Third Annual Conference of the Aerosol Society, West Bromwich p. 201 (1989)
16. J.N. Pritchard, J.J. McAughey and A. Black, J. Aerosol Sci., 19, 715-724 (1988)
17. J.J. McAughey, J.N. Pritchard and A. Black, in Present and future of indoor air quality, C.J. Bieva, Y. Courtois and M. Govaerts, eds., Elsevier, Amsterdam p. 161 (1989)
18. A. Morgan, A. Black and D.R. Belcher, Ann. Occup. Hyg., 13, 219-233 (1970)
19. A.C. Chamberlain, Proc. R. Soc. London B, 224, 149-182 (1985)

302

2023380579

**2023380580**